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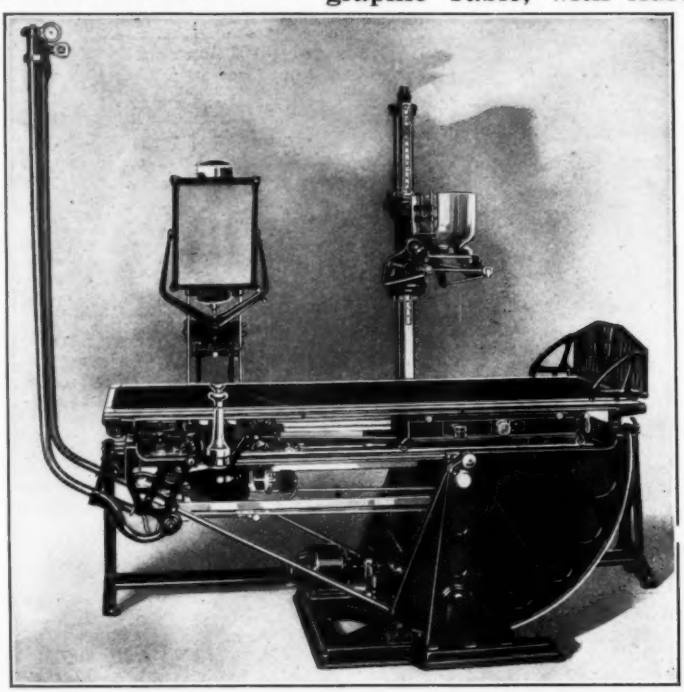

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BACTERIOLOGY OF ACUTE RHEUMATIC FEVER WITH AN EXPERIMENTAL BASIS IN ANIMALS FOR VACCINE THERAPY*

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Minneapolis

TWO aspects of the bacteriology of acute rheumatic fever are considered in this paper: (1) the evidence that acute rheumatic fever is due to a streptococcus (not a specific strain); and (2) experiments in animals suggesting a possible basis for vaccine therapy against these streptococci.

It is now generally agreed that acute rheumatic fever is an infectious disease, but opinion is still divided as to whether the disease is of streptococcic origin or is produced by an unknown virus.

The evidence that rheumatic fever is caused by a diplococcus (streptococcus) has recently become fairly convincing. The following facts support this idea: (1) streptococci can be cultured from the blood and joint fluids in a relatively high percentage of cases; (2) agglutinins against streptococci are commonly found in the blood of patients with rheumatic fever; (3) bacterial endocarditis, which is known to be of streptococcic origin, cannot be sharply separated clinically or anatomically from acute rheumatic endocarditis; (4) streptococcic lesions produced experimentally in animals (endocarditis, Aschoff nodules, subcutaneous rheumatic nodules) bear a close resemblance to corresponding lesions in man; (5) there is a definite similarity in the allergic reactions to streptococci in persons having acute rheumatic fever and in animals made hypersensitive to streptococci experimentally.

It is to be observed in Table 1 that the organism commonly recovered by the various workers from the blood, joints, and pericardial exudate before death or at the time of necropsy from patients with acute rheumatic fever or chorea is a diplococcus or a streptococcus. The frequency with which this organism is found appears to be too great to be due to secondary invasion.

The following workers have made attempts to culture the blood, joints, or pericardial exudates: Tribolet and Coyon,[1] Apert,[2] Westphal, Wassermann and Malkoff,[3] Poynton and Paine,[4] Philipp,[5] Loeb,[6] Beaton and Walker,[7] Beattie,[8] Camisca,[9] Beattie and Yates,[10] Collins,[11] Rosenow,[12] Herry,[13] La Fetra,[14] Morse and Floyd,[15] Swift and Kinsella,[16] Quigley,[17] Zinsser and Yu,[18] Suranyi and Forro,[19] Nye and Seegal,[20] and Cecil.[21] All but three of these reported finding diplococci or streptococci in from 8 to 83.5 per cent of the cases examined. Six hundred and thirty-three cases were cultured with an average of 60 per cent of positive results.

The organisms do not conform to a specific strain in respect to the action on the blood agar plate, the fermentation test, or the immunological reactions. Most of the strains reported appear to have been in what is now called the *Streptococcus viridans* group, but typical hemolytic strains in smaller numbers have been reported. Strains inactive on the blood agar plate have been mentioned. It has been our experience that these strains after a few transfers on blood agar produce the green zone about the colony. The lack of conformity to a single strain is illustrated by agglutinating and cross agglutinating strains from acute rheumatic fever and subacute bacterial endocarditis against their respective sera. The majority of the organisms from both sources fall into a common group. The remainder appear to be an irregular group made up of strains

*Read before the annual meeting of the Minnesota State Medical Association, Duluth, July 15, 1930.

TABLE I

BACTERIAL FINDINGS IN ACUTE RHEUMATIC FEVER

Authors	Time	Organism	No. of cases	Percentage positive
Triboulet & Coyon	1897	Diplococcus	6	100
Triboulet & Coyon	1898	Diplococcus	11	100
Apert	1898	Diplococcus	2	50
Westphal, Wassermann & Malkoff	1899	Streptococcus	1	100
Poynton & Paine	1900	Diplococcus	8	100
Philipp	1903		31	0
Loeb	1908	Diplococcus	45	17.5
Beaton & Walker	1903	Diplococcus	15	100
Walker & Ryffel	1908	Diplococcus	3	0
Camisa	1910	Streptococcus	9	67
Beattie & Yates	1913	Streptococcus	106	45
Collins	1913	Streptococcus	2	100
Rosenow	1913	Streptococcus	8	87.5
Rosenow	1914	Streptococcus	7	57
Herry	1914	Diplococcus	60	71.6
La Fetra	1915	Streptococcus	2	100
Morse & Floyd	1916	Streptococcus	26	8
Swift & Kinsella	1917	Streptococcus	58	12
Quigley	1918	Streptococcus	21	50
Zinsser & Yu	1928	Streptococcus	4	100
Suranyi & Forro	1928	Streptococcus	25	68
Cecil	1928	Streptococcus	29	31
Nye & Seegal	1929		25	0
Cecil	1929	Streptococcus	29	83.9
Clawson	1930	Streptococcus	100	50
Total			633	60

which, as a rule, are agglutinated only with homologous sera.

The conclusions to be drawn from the findings of the various workers on the basis of morphologic, cultural, and immunological characteristics is that the group of streptococci isolated from cases of rheumatic fever cannot be considered a specific one, but that it represents a heterogeneous group, generally green-producers on blood agar, with moderately low virulence.

Relatively little work has been done to determine the incidence of immune bodies in the sera of rheumatic fever patients. In the sera of seven of twelve cases, Tunnicliff found agglutinins for streptococci. Swift and Kinsella found no agglutinins in five patients tested. I have tested the sera of twenty-five patients having acute rheumatic fever and in twenty of these agglutinins have been present in dilutions ranging from 1:50 to 1:800 (Table 2).

The distinction between subacute bacterial endocarditis and rheumatic endocarditis is often difficult to make clinically. Streptococci may be cultured from the blood in both cases, but much more readily from bacterial endocarditis because of the greater number of organisms in the blood stream. A consideration of the method of culturing may be helpful in deciding whether the organisms cultured are from a case of acute rheumatic fever or from a case of bacterial endocarditis. Growth in liquid media may be from either, but, if several colonies develop from a blood agar plate, when the blood used is from the patient, the evidence is strong that the case is what is called clinically bacterial endocarditis.

In endocarditis immediately associated with rheumatic fever (rheumatic endocarditis, simple verrucous endocarditis), the heart valves on gross examination show small, smooth, globular nodules at the points of contact of the leaflets. The chief lesions found on the valves in subacute bacterial endocarditis are soft villous vegetations. The consistence and size of these vegetations make it easily possible for particles to break loose into the blood stream and produce the embolic processes so commonly found in subacute bac-

TABLE II

FREQUENCY OF IMMUNE BODIES IN PATIENT'S SERUM

Author	No. examined	Positive	Negative
Tunnicliff	12	7	5
Swift and Kinsella	5	—	1
Clawson	25	20	5

terial endocarditis, but vegetations of the rheumatic type are found mixed in with the large soft vegetations in at least two-thirds of the cases of subacute bacterial endocarditis. This frequency of association of the two lesions seems to be too great to be explained on a basis of double infection.

Microscopically the lesions of the valves in acute rheumatic endocarditis show the inflammation beginning within the substance of the leaflet. The inflammatory reaction is polyblastic in character. The cells are round and spindle shaped with abundant cytoplasm. The valve tissue beneath the thrombus in cases of subacute bacterial endocarditis shows a reaction similar to that found in the valves in acute rheumatic endocarditis. This gross and microscopic similarity in the vegetations and in the cellular reaction strongly suggests a similar etiologic agent in the two kinds of endocarditis. The subacute bacterial type has been definitely proved to be of streptococcic origin.

In considering whether the streptococcus may be the etiologic agent in producing acute rheumatic fever, much importance rests upon whether lesions similar to human rheumatic lesions can be produced experimentally in animals. Many workers have produced valvular endocarditis experimentally by injecting streptococci. Apparently, in most cases the lesions are large and simulate the bacterial type, but vegetations characteristic of the rheumatic type are not infrequently encountered. Experimental nodular lesions structurally similar to the so-called Aschoff bodies have been produced in the myocardium and the subcutaneous tissues by Coombs, Miller and Kettle, Jackson, and me. By injecting streptococci into rabbits and monkeys I was able to produce an arteritis which appears similar to that described by Pappenheimer and Von Glahn as being specific for acute rheumatic fever.

The experiments and observations of Swift and Birkhaug and my recent experiments have shown that there is a marked similarity in the hypersensitiveness (allergy) in patients having acute rheumatic fever and in animals made hypersensitive experimentally to streptococci.

The facts that streptococci are recovered from the blood and joints in so high a percentage of cases of acute rheumatic fever, that agglutinins for streptococci are so frequently found in the blood in cases of acute rheumatic fever, that the lesions in acute rheumatic fever are so similar to those in subacute bacterial endocarditis (which is known to be due to streptococci), that lesions similar to those found in acute rheumatic fever can be produced in animals by injecting streptococci, and that there is a definite similarity in the hypersensitiveness to streptococci strongly suggest that the most probable cause of acute rheumatic fever is the streptococcus. It remains to be seen what can be done in the way of treatment by combating these organisms.

In recent experiments I have been able, by giving intravenous vaccinations, to desensitize animals made hypersensitive to streptococci experimentally and prevent the development of subcutaneous nodular lesions, which are the nearest approach to rheumatic lesions in animals yet produced. Subcutaneous injections of vaccines do not protect the animals. Since the hypersensitiveness to streptococci in patients with acute rheumatic fever appears to be similar to the hypersensitiveness in these animals, it seems possible that patients with acute rheumatic fever might be desensitized and the further progress of lesions prevented by receiving intravenous streptococcic vaccinations.

CONCLUSIONS

1. Acute rheumatic fever appears to be due to an infection with streptococci.
2. These streptococci do not appear to be a specific strain.
3. Streptococcic rheumatoid lesions in animals made hypersensitive to streptococci can be prevented by intravenous vaccination.
4. This method of vaccination may be indicated in patients with acute rheumatic fever, since such patients are hypersensitive to streptococci.

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AMBRUSTER, RUSBY—AND ERGOT

For almost three years one Howard W. Ambruster, an importer of ergot, with the advice of H. H. Rusby, has charged vociferously, repeatedly, frantically, that the fluidextract of ergot U. S. P. of American manufacture, was grossly substandard. Controlled scientific evidence from creditable sources to sustain the charges has not been presented. In innumerable circular letters, in broadsides, in statements to the press and wherever he can get a hearing, Mr. Ambruster asserts there is a conspiracy on the part of the American Medical Association and of the Food, Drug and Insecticide Administration of the U. S. Department of Agriculture to approve substandard drugs. Not only has the American Medical Association as an organization been attacked, but various officials have been subjected to Ambruster's diatribes. Moreover, the American Pharmaceutical Association, the office of the Secretary of the Treasury, many pharmaceutic houses, office of the Secretary of Agriculture, the U. S. P. Revision Committee and others have been included. The campaign which Ambruster has waged and continues to wage would command admiration if there appeared to be any sincerely altruistic motive for it. But there appears to be no evidence to substantiate his charges. Both the Council on Pharmacy and Chemistry and the Journal of the American Medical Association have reported on two different occasions that there was no evidence that fluidextract of ergot U. S. P. as found on the market should not be used with confidence by American physicians. Last June the Committee on Agriculture of the United States Senate held hearings on the Ambruster charges. The evidence permitted to go into the record was not limited to the "indictment." Members of the medical profession and officials of the American Medical Association were grossly accused by Ambruster and Rusby. While a definite report has not been made by the Senate Committee as a whole, the impression created on the technical members of the committee and apparently shared by most of the non-technical members was fairly well summarized by comments made by Senators Copeland and Hatfield in the closing days of the hearing. Senator Copeland held that no evidence has been presented to show that the quality of fluidextract of ergot was a menace to mothers in childbirth and that he had not heard any testimony to show that the government officials had been lax in their enforcement of the law. (Jour. A. M. A., September 6, 1930, p. 722.)

RHEUMATIC FEVER IN CHILDHOOD*

HENRY F. HELMHOLZ, M.D.,
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RHEUMATIC fever in childhood is an acute infectious disease, probably caused by a streptococcus and characterized by proliferative and exudative changes in the joints, serous membranes, heart, blood vessels and skin. The so-called rheumatic cycle includes tonsillitis, arthritis, carditis, and chorea. The disease, named for its most striking symptoms, is serious largely because of its crippling effect on the heart. Although the involvement of the joints is acute and severe, they usually become completely normal after an attack.

ETIOLOGY

According to practically all statistics, the disease is more common in the poorer classes in the United States as well as in Europe. The incidence of the disease has decreased with improvement in general hygienic conditions. Rheumatic fever makes up from 3 to 7 per cent of the medical diseases observed in children's hospitals.

Season.—In the United States and in Europe the majority of cases are noted in the winter and spring. Relapses also are most common at these seasons. In England, on the other hand, more cases are noted in the autumn. This difference in incidence never has been adequately explained.

Familial relationship.—There appears to be definite familial relationship to the disease. Whether this is due to environment or to infection carried from parent to child, or whether there is a real predisposition is difficult to determine.

Age.—The disease is seen only infrequently in children less than five or six years of age. In a series of 2,051 cases, Curschmann and Eckstein found that the disease had been acquired before the fifteenth year in 7.6 per cent, and of these children 80 per cent had carditis.

PATHOLOGY

The pathologic changes in the joints are not typical. Characteristic changes of rheumatic fever are found in the myocardium in the so-called Aschoff body, a small group of lymphocytes with a central mass of multinucleo-epithelioid cells. In recent years it has been shown that not only the heart, pericardium, myocardium, and endocardium are often affected but also the entire circulatory system, both pulmonary and systemic. The lesions of the heart probably are embolic in nature, the endocardial lesions resulting over areas of necrosis due to small emboli in the vessels of the valves. Recently, the interstitial changes of the mitral valves involving the entire leaflet have been emphasized. The changes in chorea probably are vascular.

SYMPTOMS

There is usually an antecedent upper respiratory tract infection, at times strikingly associated with fatigue, followed at a varying interval by high fever, marked perspiration, increased pulse rate and arthritis. Pain, tenderness, redness, and swelling in one or more joints appear, with later involvement of others, principally of the large joints. There may be a single cycle or a series of cycles of fever and arthritis involving numerous joints.

Characteristic arthritis disappears without permanent injury to joints. In certain cases the upper respiratory tract infection and the arthritis alone are manifested. In a great many cases, however, there also is evidence of involvement of the heart. In 95 per cent of Swift's series transitory changes of the heart according to weekly electrocardiograms were shown.

Carditis.—As the involvement of the heart is the most serious aspect of rheumatic fever, it is important to consider each case of rheumatic fever as possibly carditis until it has been proved definitely that the heart is not involved. The presence of Aschoff bodies in practically all cases of rheumatic fever demonstrates that myocardial changes are constant. The incidence of endocarditis is lower and that of pericarditis is still lower.

Since functional murmurs occur in childhood in practically all febrile conditions, it is at times extremely difficult to determine organic disease of the heart. It is practically never possible to make a diagnosis of endocarditis from a single examination of the heart alone. I have found

*From the Section on Pediatrics, The Mayo Clinic, Rochester, Minnesota. Read before the Minnesota State Medical Association, Duluth, July 15, 1930.

the following points useful in the differential diagnosis: the functional murmur tends to be basal, but may be present at the apex in maximal intensity; it is usually rather harsh, representing a fraying out of the first sound without a sharp beginning but generally with a sharp end-point. The murmur tends to be louder in the recumbent position and to disappear on exercise. The murmur of endocarditis in its early stages is heard as a soft blowing, with a sharp beginning and a sharp end-point, and it is distinct from the first sound. The murmur is the same whether the patient is lying or standing and becomes more intense with increased action of the heart. In exceptional cases I have noted functional murmurs gradually assume the characteristics of an organic murmur.

A sufficient number of cases of acute endocarditis of the mitral valves has been described to demonstrate that the lesion may heal completely. Amberg and Willius have described such cases seen at The Mayo Clinic.

Pericarditis is usually manifested by a to-and-fro murmur, generally heard best along the left border close to the sternum.

Nodules.—The presence of rheumatic nodules seems to vary considerably from year to year. It has been my experience that during certain periods rheumatic nodules are very common; at other times they are not found, or are only present in small numbers. They are usually found on the back of the head or around the large and small joints of the feet, hands, legs and arms. By flexion and tightening of the skin over the joints the nodules usually appear about 0.5 cm. in diameter, although they may be as large as 1 cm. These nodules are helpful in the diagnosis of rheumatic fever and are of serious prognostic import if present in large numbers and in new crops.

Chorea.—Chorea should definitely be considered as part of rheumatic fever and not as a complication occurring as a later manifestation of the disease. Not infrequently it is a primary mode of onset.

Growing pains.—This symptom as evidence of rheumatic fever is extremely difficult to evaluate. The relation of growing pains to the disease has been over-emphasized by so many observers that indefinite pains in the lower extremities have been considered as a definite manifestation of rheumatic fever. In my experience, a great many cases of growing pains are the result of flat feet, and can probably be explained on this basis. If an acute infection of the upper respiratory tract is followed by indefinite pains in the lower extremities the possibility of rheumatic fever should be considered.

DIAGNOSIS

Polyarthritis that responds to treatment with salicylates and disappears without residue justifies a diagnosis of rheumatic fever. The occurrence of subcutaneous nodules, cardiac involvement, and chorea, alone or in any combination, is diagnostic of the disease.

The differential diagnosis must take into account polyarthritis disappearing without residue that occasionally is seen in serum disease, polyarthritis of gonorrheal origin, and polyarthritis of lupus erythematosus disseminata. Polyarthritis occasionally is seen with chronic ulcerative colitis. Acute rheumatic fever of the spine may be taken for meningitis because of the positive Kernig's sign and stiffness of the neck. The absence of meningeal irritation on spinal puncture, however, tends to make the diagnosis clear. Osteomyelitis may occasionally present diagnostic difficulties.

PROGNOSIS

The death rate from acute rheumatic fever is between 3 and 5 per cent. The prognosis becomes increasingly bad with each relapse because of the increasing frequency of endocarditis, myocarditis and pericarditis. In cases of obliterative pericarditis the prognosis is especially poor, although recent operative procedures have improved it remarkably. The presence of fever and an increased leukocyte count, even after the disappearance of other symptoms, usually means persistent infection. Elimination of foci of infection and a period of rest sufficiently long to give the cardiac lesions an opportunity to heal improve the prognosis. Kaiser, in a recent report on the effect of tonsillectomy on the incidence of rheumatic fever, stated that removal of the tonsils definitely decreased the incidence of rheumatic fever and carditis but that the number of cases of chorea had increased.

TREATMENT

It is well to emphasize the beneficial effect, almost forgotten, of salicylates given in large doses of from 4 to 6 gm. a day during the acute stage,

watching carefully for evidence of toxicity, nausea, vomiting, and nephritis. Smaller doses of the drug may be continued for from one to two weeks after the disappearance of symptoms.

Removal of all foci of infection, tonsils in particular, should be carried out as early as possible after the disappearance of acute symptoms. This should be followed by a long period of rest, varying from six months to a year, in an attempt to effect cure of the carditis and chorea. As it is not possible to determine the changes in the heart during life, such protracted periods of rest seem to be justified.

SUMMARY

Rheumatic fever in childhood may be monosymptomatic or may show a few or all of the manifestations of the so-called rheumatic cycle. Complicating disorders of the heart are very common and constitute the serious danger of the infection. All patients with rheumatic fever should have foci of infection removed, and should be given a period of from one to two months of restricted regimen. Patients with carditis or chorea should be kept at absolute rest for periods of from six months to a year, after which the normal mode of life may be resumed gradually.

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INCREASED POTENCY OF VIOSTEROL PREPARATIONS

The Wisconsin Alumni Research Foundation informed the Council on Pharmacy and Chemistry that the accumulated clinical experience with viosterol has shown that better results in the treatment of rickets are secured when a dosage of Vitamin D is used larger than that originally recommended and that the maximum limits of safety as to the amount of Vitamin D that can be used has now been more definitely determined than was the case when preparations of viosterol were first put on the market. Instead of increasing the dosage of the present products, the Foundation and its licensees determined to increase the potency of the preparations. It was decided to increase the potency of viosterol in oil so that instead of having 100 times the Vitamin D potency of a standard cod liver oil as determined on rats by the Steenbock line test, it shall have 250 times that potency, and, provided the Council should agree, to increase the potency of cod liver oil with Viosterol so that instead of having 5 times the potency of a standard cod liver oil as determined on rats by the Steenbock line test, it shall have 10 times that potency. The Foundation announced that these preparations of increased potency would be placed on the market beginning with October first. The Council on Pharmacy and Chemistry announces that it has accepted the changes of potency determined on by the Foundation and has changed the name of viosterol in oil 100 D to viosterol in oil 250 D and the name of cod liver oil with viosterol 5 D to cod liver oil with viosterol 10 D and has continued the acceptance of the viosterol preparations already accepted, under the new names. (Jour. A. M. A., October 4, 1930, p. 1021.)

RHEUMATIC CARDITIS*

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IT IS generally accepted that acute rheumatic fever is an infection due to some type of streptococcus modified by predisposing factors the nature of which we know very little.

The older term, acute articular rheumatism, stressed the joint manifestations. The more recently accepted designation—acute rheumatic fever—indicates its more general distribution. Even this name is not altogether correct, as there has accumulated much evidence to show the necessity of considering this disease to be of a subacute or chronic nature.

It is essentially a perivascular disease distributed throughout the body in a manner that has been likened to the wide-spread distribution of tuberculosis and syphilis. The various tissues of the heart are especially prone to involvement, and the resulting carditis is a very essential part of the picture. This form of heart disease must not be considered as being secondary to, or resulting from joint inflammation. It often appears without the presence or history of any arthritis. Although the joint lesions are the most dramatic in their manifestations, the heart lesion is most important, and may persist for many years after the arthritis has disappeared.

Heart disease ranks first in the list of those diseases producing death. Next to hypertension comes acute rheumatic fever as the agent responsible for heart fatalities. It has been estimated that rheumatic carditis contributes over 20 per cent of all cardiac deaths. The importance of this figure is further added to by the fact that most of this group are infected early in life, and the age group of these deaths is much earlier than the group caused by hypertension and arteriosclerosis.

It is not my purpose to discuss in detail the etiological factors concerned in the production of this disease. The relationship of foci of infection and rheumatic carditis has not been fully established. Hölsti's investigations have shown that the inflamed tonsil in this disease may be merely a part of the acute rheumatic fever, and not the source of the infection.

There also appears to be some hereditary or constitutional factors that predispose to the infection. Repeatedly, cases of rheumatic carditis are found in different generations of the same family. This has occurred sufficiently often to impress students of this disease with the possibility of there being an inherited predisposition.

PATHOLOGY

In the acute stages the disease is a septicemia. The lesions are formed around the blood vessels throughout the body, but especially in the joints, heart, nervous tissues and skin. The histology of the pathological change is essentially the same, no matter what tissue is involved. In the heart the acute lesion consists of a necrotic center with a zone of leukocytic infiltration and cell proliferation. Many round cells are present, and larger multinucleated cells are prominent. These lesions are all located around and in the walls of the smaller blood vessels of the coronary circulation, and bear the name Aschoff nodules.

Some degree of heart involvement is found in almost every case. Proliferative and exudative lesions are found in the endocardium, myocardium and pericardium. These tissues may be involved separately or together. It is not certain whether these changes are produced by the direct action of the bacteria or the toxins of the disease, or whether they are of the nature of an allergic reaction. Active lesions may be present for as many as thirty years after the disappearance of the joint inflammation. Scars in the myocardium are not frequently found, indicating the transient nature of most of these lesions.

The conduction system is especially liable to injury. Although this is difficult to demonstrate histologically, it is well shown by electrocardiograph studies. Poyton, who has studied this disease for over twenty-five years, considers the myocardial lesion to be the most frequent and most important change in children. It is usually overlooked for the reason that it often produces no clinical evidence.

Although the myocardial changes are the most important in the acute stages, it is the valvular disease that produces the severe crippling in later

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life. The valves most commonly involved are the mitral and aortic. Stenosis of the mitral valve is the typical valvular deformity found in this condition.

For many years it has been held that the valvulitis occurred from the blood in the heart chambers, the inflammation extending from the surface inwardly. The more recent conception considers the infection starting as an interstitial valvulitis, and extending toward the surface. This belief has been further substantiated by the discovery of capillaries in the heart valves in adults. It was thought that these capillaries, normally present in the fetus, disappeared at birth. Deep valvulitis, without superficial erosion, is frequently found, and especially often in the valves of the right heart. Superficial valvulitis rarely occurs alone. We should consider the valvular lesion not as an endocarditis, but as an interstitial valvulitis.

The early valvular lesion consists of proliferation and exudation, with the edema playing an important part. The contact of the swollen leaflets at the margin of closure, very likely contributes to the superficial changes that take place in the approximating edges of the valves. In some individuals the proliferative changes are more marked than the exudative. This type of change is capable of producing the more severe kind of valvular deformities. Although valvular disease may be present at the period of acute inflammation, its evident signs may not appear until contraction of the scar occurs.

Pericarditis is a fairly frequent occurrence. The subpericardial tissue is richly supplied with blood vessels, and it is around and in these blood vessels that the inflammation takes place. If the process is sufficiently intense, the pericardial surface will be involved, and the characteristic rub will be produced. Severe congestion in the subpericardial capillaries may cause a rupture through into the pericardial sac, and a hemorrhagic pericarditis results. Exudation of fluid may take place, and often a large effusion is present.

The lesions in the heart take place in and around the smaller ramifications of the coronary arteries. Occasionally some of the larger blood vessels are involved, and anginal pains may be produced.

There may be some question as to the advisability of including the subject of subacute bacterial endocarditis in the discussion of rheumatic carditis. The exact relationship between these two forms of heart disease is not entirely clear. They present several points of similarity that would suggest that they are essentially the same disease. The valvular lesion, however, is very different. In the subacute disease the valve leaflets are injured, with the production of a soft, friable vegetative growth that breaks off and produces the embolic phenomena that are so characteristic of this type of valvulitis. The lesions in acute rheumatic fever are smooth and firm, and cannot be dislodged. Subacute bacterial endocarditis is a fatal disease, and about one-half the cases that come to autopsy show some of the typical rheumatic lesions, in addition to the vegetative changes found in the valves. Many of these patients have a history of previous joint lesions, subcutaneous nodes, and rheumatic carditis. In both conditions the blood will yield cultures of the same type of streptococcus. This would suggest that subacute bacterial endocarditis is nothing more than a very severe form of rheumatic heart disease, brought about by an unusually virulent type of streptococcus, or occurring in an individual who is extremely sensitive to this infection. It is important to make the differentiation, because of the difference in prognosis.

THE ONSET OF RHEUMATIC CARDITIS

The development of rheumatic carditis cannot be described as occurring in any typical manner. It is an essential part of the disease and has no time relationship to the appearance of the other lesions.

In children there may be present no findings other than the mild joint pains, growing pains, or slight chorea, which may be so insignificant as to be completely overlooked. In adults the joint and throat inflammation is usually quite definite, and demands a great deal of attention. The subcutaneous nodes, when they are present, are usually accompanied by heart involvement.

Leukocytosis and fever are the two main findings indicating activity. Often the valvular lesion does not present the diagnostic physical findings, for some time after the acute symptoms have disappeared. In fact, it is usually impossible during the acute stage to definitely say that a valvulitis is present.

Even very slight evidence of the presence of acute rheumatic fever, such as very minor joint

pains, an occasional subcutaneous node, low grade fever and leukocytosis, should make one suspect the presence of a heart lesion.

It must be emphasized that there are a number of cases of rheumatic carditis that do not at any time present any other symptoms of acute rheumatic fever. This group comprises from 20 to 30 per cent of all the cases of rheumatic heart disease.

PHYSICAL FINDINGS AND DIAGNOSES

There is often a great deal of difficulty in determining the presence of the heart lesion. This is especially true when only the myocardium is involved. There are several observations that can be made, aside from the heart examination. Any increase in temperature or leukocytosis in a patient showing joint symptoms, but without the involvement of new joints, points toward the onset of a carditis. The presence of subcutaneous nodes usually indicates that the heart has been damaged. An unusually rapid pulse, out of proportion to the temperature, frequently means a myocardial injury.

The physical signs of early myocardial and endocardial involvement are usually not very definite. Dilatation can occasionally be made out by noting the shifting of the left border to the outside of the left mid-clavicular line. When the conduction system is involved, various degrees of heart block may be present, although it requires the use of an electrocardiograph to determine the less severe changes in the conduction time, and the other myocardial lesions. In the more severe degrees of heart block, dropped beat can be made out.

During auscultation, muffling of the apical tones is said to indicate edema of the valves. Frequently a soft systolic murmur is heard along the left sternal border. Libman believes this is a very early sign of valvular involvement. Ordinarily not much significance is attached to a murmur of this type, but when it occurs during the course of a rheumatic fever it may indicate cardiac damage, especially if it is accompanied by an accentuated second pulmonic tone.

When the pericardium is involved, and the surface becomes roughened, a characteristic sound is produced. This is usually a to-and-fro murmur, rather rough, and heard close to the ear. When the visceral and parietal pericardium become adherent this rub disappears. The disappearance and reappearance of the pericardial murmur indicates that activity is still present, and it is a valuable sign. In practically every case the presence of pericarditis means also some involvement of the other tissues of the heart.

Precordial pain and cutaneous hypersensitiveness often indicate the presence of a myocardial lesion. Swift has shown that over 70 per cent of the cases with arthritis and carditis had precordial pain, and that 90 per cent of the cases with subcutaneous nodules had pain and hyperesthesia.

He also observed that those patients with precordial distress had, as a rule, a more severe form of heart disease. The heart muscle itself does not respond to stimuli with pain, but the blood vessels and especially the adventitia, are capable of producing pain and hyperesthesia when they are inflamed. In acute rheumatic fever the lesion is located in and around the adventitia. If the large coronary vessels are involved the distress may be very similar to that which occurs in coronary sclerosis. The pericardial inflammation also produces precordial pain. Sometimes this is very severe, and often occurs with respiration.

THERAPY

Unfortunately we have no specific treatment for rheumatic carditis. From our knowledge of this disease, it appears that we must look to the immunologist for help in perfecting a desensitizing or immunizing agent.

Drugs do not play a prominent part in the treatment of this condition. There is no good evidence to show that salicylates have any effect in preventing the heart lesion or modifying the course of the carditis. There is no question but that it is of great value in relieving the patient of the distress due to the joint inflammation and fever. However, the remarkable symptomatic relief that it frequently produces, often gives the physician a false sense of security, as it masks the onset of the heart lesion.

Digitalis is frequently given as soon as a heart lesion is suspected. This form of therapy is certainly not indicated unless there are definite signs of heart failure. It is toxic to the heart muscle which is already suffering from the effect of the toxin of rheumatic fever.

The nature of the pathological change that takes place in this disease indicates the necessity for resting the heart until it has completely recovered from the effect of the acute process. As long as the pulse is rapid there is a good possi-

bility that activity is still present. Fever, even though it be of a very low grade, is significant, and, along with leukocytosis, indicates that the infection is still present. These three conditions should be watched carefully. It is difficult to say just how long rest should be enforced. Four to six weeks after all signs of activity have left should be a minimum period of quiet. Usually the heart will completely recover from the myocardial lesion if given enough time. Too early or too vigorous exertion may cause serious damage.

It has been suggested that these heart patients should be treated in a manner similar to the treatment of a tuberculous individual—rest during the acute periods, followed by graduated exercise, sunlight and fresh air, until the patient is able to again become useful and active. Much work along these lines is now being done, especially in England, with rather encouraging results.

Lastly there arises the question—what part do foci of infection play in this disease, and how should they be attacked? As a matter of principle, all areas of infection should be removed. Infected teeth and sinuses do not seem to have a close relationship to this disease. Inflammation of the tonsils and acute rheumatic fever occur together so frequently that it would seem that there is something more than a mere coincidence. However, it has not been definitely shown that removal of tonsils prevents the rheumatic carditis or changes the course of the disease very markedly. It is generally agreed that infected tonsils should be removed, but it should not be done during the acute period of the disease, but rather after the fever has subsided.

A guarded prognosis should be given in every case, remembering that evidence of serious heart trouble may appear only many months after the first attack of arthritis; that this is, frequently, a very chronic type of disease, accompanied by many relapses or reinfections; and that with each exacerbation the heart lesion may become progressively worse. It should also be borne in mind that rheumatic carditis frequently occurs without any of the other obvious signs of acute rheumatic fever. Complete rest for a sufficient length of time, followed by graduated exercise, is the most important therapeutic measure.

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THE PROTEAN PATHOLOGICAL MANIFESTATIONS OF RHEUMATIC FEVER*

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THE modern conception of the pathology of rheumatic fever is rapidly undergoing a change. The most evident clinical symptoms of the disease and the studies of the pathologists of twenty-five years ago had directed our attention toward the joint signs and the circulatory "complications." Aschoff in 1904 described in detail the histopathology of the minute inflammatory nodules of the ventricular myocardium, and Clawson has recently critically reviewed our present knowledge of the "Aschoff nodule" as observed in man and similar lesions as produced experimentally in animals. During the past decade, but especially during the past five years, there has been a renewed interest in the study of the histogenesis of this lesion which was formerly considered specific for this disease.

The "Aschoff nodule" is usually found in close relation to small arterioles or capillaries, and represents the reaction of connective tissue to the virus or its toxic products. The "typical" nodule varies somewhat depending upon the tissue involved, the virulence of the infection and the resistance of the patient. A constitutional factor has been suggested. In general the tissues show both a proliferative and an exudative reaction of varying degrees; it may be essentially proliferative with the formation of multinuclear giant cells resembling other types of granulomata, or it may be essentially exudative. The latter may be mostly lymphocytic and plasma cells, or at the other extreme be essentially polymorphonuclear with microscopic abscess formation, but suppurative lesions as a rule do not occur because of the low virulence of the rheumatic infection.

A lesion close to a serous membrane such as the epicardium, pleura or joint synovial membrane is likely to excite an exudative process in the serous cavity.

The late lesion or reparative scar is just as variable. The specificity of the "Aschoff nodule" has more recently been questioned, since an atypical proliferative nodule may be found in some "non-rheumatic" conditions where the virulence is low. It has been described for the myocardium in scarlet fever, pneumonia, subacute bacterial endocarditis, meningococcic infections, erysipelas and in general where the infection is due to relatively avirulent sterptococci.

The relation of allergy to the production of some of the tissue reactions, similar to that which is believed to occur in tuberculosis, is at the present time a very attractive field for study. It is difficult to explain all of the immune reactions of this disease without assuming such an hypothesis. Some experimental studies afford considerable evidence in favor of this viewpoint.

In another paper of this symposium the cardiac lesions of rheumatic fever have been discussed but an additional study of all of the known lesions affords us a better conception of the pathological process.

A. Lesions involving the larger arteries.

This is supposedly by the path of the vasa vasorium.

(1) The aorta.

Klotz in 1912 described a non-suppurative mesaortitis and also an involvement of the adventitia. Intimal changes may also occur, often similar to the endocardial lesions of the auricle. Typical Aschoff nodules are sometimes present near the nutrient vessels and in the adventitia. During the healing there may be patchy destruction of muscle fibres in the media due to interference with the blood supply. A patchy aortitis results which is probably in the more extreme cases a cause of dissecting aneurysms. The large areas of dilatation which occur in syphilic aortitis generally do not occur because there is not the widespread destruction in rheumatic disease of the aorta.

It is well to remember that not all cases of aortitis are syphilitic.

(2) The main pulmonary arteries may show lesions similar to those in the aorta, namely a proliferative mesarteritis near the nutrient vessel with secondary changes in the adventitia and intima. Thrombi may form in the injured vessels which may cause additional damage such as pul-

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monary atelectasis and compression endarteritis. The symptom-complex known as Ayerza's disease may be the end-results of such an arteritis.

(3) Lesions of other large arteries have been described in patients dying a cardiac death, but the evidence leaves a doubt as to the real etiology in many of these reports.

B. Lesions primarily affecting the smaller arterioles and capillaries.

As a rule the moderate size arteries show little or no demonstrable lesion. Only arterioles the size of an arterial nutrient vessel or smaller seem to be involved.

(1) The myocardium, valve leaflets, subendocardium, and subendothelial capillaries of the pericardium may be affected. These lesions have been discussed by others in this symposium.

(2) Pulmonary Aschoff nodules involving the interstitial tissue occasionally occur. This may be followed by an exudative process into the alveoli with some of the signs of an atypical pneumonia. It does not become a suppurative or an essentially exudative process such as clinicians see in lobar or post-influenzal pneumonias. The patients are often so free from symptoms and signs that true rheumatic pneumonia is overlooked. It is rarely in itself fatal, but there may be crippling sequelæ due to scar formation.

Rheumatic pneumonia must be differentiated from a lobar or bronchial pneumonia, complicating acute rehumatic fever or its later complications, and it should not be confused with the embolic process observed in sub-acute bacterial endocarditis. Compression of the lower part of the left lung associated with pericardial effusion may show many of the signs of a pneumonia.

Pleuritis accompanies pneumonia or pericarditis, or it may be the only demonstrable chest lesion. It may be fibrinous or may be accompanied by an effusion. Many of the "wet" pleurisies diagnosed in the past as tuberculous were probably due to "masked rheumatic infections" in the broader sense of the term.

(3) Rheumatic involvement of the other internal organs has not so commonly been observed, but now and then many of the organs may show areas of rheumatic proliferation or typical nodules. Clinical kidney damage is rare, but occasionally lesions of the glomerular vessels have been described in necropsy material. In chorea there are commonly cerebral perivascular lesions resembling the rheumatic nodule.

(4) The typical rheumatic tissue reaction has commonly been found in the joints, subcutaneous tissues, tendons, muscles and other connective tissues. In general it seems that all capillaries of a certain size may be affected. There has been considerable speculation as to the relation of the rheumatic infection to chronic arterial disease.

In conclusion we may state that the pathological process in acute rheumatic fever is a non-suppurative, proliferative or exudative lesion about the smaller arterioles and capillaries throughout the body. The symptoms and signs differ depending upon the number and distribution of these lesions, but most commonly our attention is directed toward those organs where the disturbance is clinically most easily demonstrable.

THE NON-ARTICULAR FORMS OF RHEUMATIC FEVER*

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AN IMPORTANT feature of rheumatic fever, and one that cannot be overstressed, is that inflammation about the joints is not necessarily an accompaniment of the infection. These non-articular manifestations are just as much a part of the infection as is the arthritis and they must be recognized as such and treated accordingly. Although the various clinical manifestations of the disease are quite different the underlying pathological lesion is the same.

The non-articular forms of rheumatic fever cannot be discussed as a whole, because they are different clinical entities and although it is possible to have one or more of these different manifestations present at the same time, usually, however, they appear singly.

Chorea frequently occurs without any other of the so-called rheumatic manifestations in children. One striking feature of chorea is the frequency with which one or another of the symptoms and signs of rheumatic fever may appear as isolated evidence of activity. It may be many years before the child shows enough different manifestations to establish the true nature of the disease. The symptoms though difficult to describe are never forgotten when once seen nor do they offer any difficulties to the diagnostician except in their rarer forms. According to the severity we recognize three forms: the mild, the severe, and that associated with delirium. It should be remembered that choreiform movements are increased by excitement or obstruction, entirely cease during sleep whether natural or induced by drugs, and that neither sensibility nor usually reflex activity is affected.

Several of the non-articular forms of rheumatic fever are capable of producing a great deal of pain, which is the complaint that brings the patient to his physician. Muscular rheumatism, fibrositis, or myalgia, terms that are used interchangeably, is an extremely common form of ailment. It is a condition of muscular fibrositis, due to microbic toxic absorption; to anoxemia through faulty local blood supply; or colloidoclastic tissue changes. It occurs frequently as a sequel to tonsillitis and is peculiarly likely to develop after exposure to cold or dampness, excessive fatigue, and any factors tending to reduce the body reserves. The condition may involve any muscle or group of muscles, but usually the lumbar muscles (lumbago), the muscles of the chest (pleurodynia), and the cervical muscles (rheumatic torticollis) are the ones affected. The fundamental diagnostic points are diffuse tenderness of the involved muscles; the pain is distinctly related to muscular contraction, and is absent or greatly relieved when the muscle is at rest. Except in the case of pleurodynia the amount of pain present is dependent chiefly upon the voluntary use of the muscle group affected. In pleurodynia the movement is obligatory, but, in general, spontaneous pain is relatively slight or wholly absent, though local tenderness may be extreme. The presence of tenderness over the muscles involved and the prompt pain response to movement make the diagnosis.

Care must be taken lest a persisting torticollis due to organic disease be mistaken for myalgia, which is ordinarily of but a few days duration. Pleurodynia must never be accepted as the diagnosis without a careful examination of the heart, lungs, and pleura. A strikingly similar pain may be present with intercostal neuralgia, but tenderness over the nerve and pain regardless of motion and definite points of maximal tenderness should clarify the diagnosis. The precursor pain of herpes zoster must also be kept in mind.

Neuritis often accompanies muscular rheumatism as a primary involvement or reflection of adjacent inflammatory processes. Like muscular rheumatism, neuritis usually follows exposure to cold, dampness, and excessive fatigue. Neuritis most commonly involves the sciatic and brachial nerves and is often hard to distinguish from myalgia, but the pain follows more closely the course of the nerve, is aching in character and the loss of function of the regions supplied may be more complete. Both muscular rheumatism and brachial neuritis must be differentiated from cervical rib, tumor of the neck,

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glands in the axillæ, aneurysm of the subclavian artery, spinal tumor and caries of the cervical vertebræ.

Subcutaneous nodes or nodules are one of the most characteristic and diagnostic lesions found in rheumatic fever. They are small, hard, tender masses, deep under the skin and frequently cause the patient a great deal of pain. The lesions may come anywhere on the body but are usually on the lower extremities. There may be only a few nodules present or there may be many, but even one or two typical lesions are sufficient for a diagnosis. The nodules are not constant and vary from day to day or may disappear for considerable periods of time.

Pleurisy is not an unexpected accompaniment of severe polyarthritis, but it must be remembered that it may be an isolated manifestation of rheumatic fever. Although the condition is rare, cases are encountered where pleurisy precedes any other signs of infection by months. This makes it extremely difficult at times to decide whether a patient is suffering from rheumatic fever or tuberculosis. Effusion is the exception rather than the rule in this type of pleurisy and the pleuritic rub tends to come and go.

Erythema nodosum, a very acute type of inflammatory infection of the skin, is characterized by the formation of various sized, roundish, more or less elevated erythematous nodes and is attended by varying degrees of systemic disturbance. The lesions are seen for the most part on the tibial surfaces and may often be limited to these regions. Not infrequently, however, other regions are involved, more especially the arms and forearms. They are rarely present in great numbers, usually from two or three to thirty being found. They begin as a rule as deep seated nodules, rapidly growing larger and becoming elevated. In size they vary from that of a cherry to a hen's egg, are rounded or oval, tender and painful, and have a glistening tense look. They are of a bright red color that merges gradually into that of the normal skin. Later the color changes to a purple and then the various color changes of an ordinary bruise. As they develop they become soft and semi-fluctuating but they never suppurate. The condition should not be confused with the lesions of erythema induratum, which are thought to be of a tuberculous nature. Another skin lesion related in some respects to erythema nodosum is that which is described as erythema multiformi. Many authors believe these two skin diseases to be closely related.

The above mentioned conditions, with the exception of chorea, are usually found in adults. In children various mild pains are frequently present that are commonly referred to as growing pains. Without question many of these children are suffering with rheumatic fever. Many investigators consider them to have the same significance as severe arthritis and that they are to be considered as indication of active rheumatic infection likely to implicate the heart. Without doubt in many instances the so-called growing pains do not indicate active rheumatic fever. The differentiation is difficult to make, but in a child with other evidence of infection they must be viewed with concern.

In conclusion it is well to emphasize again that the non-articular forms of rheumatic fever are just as important as the articular forms and challenge our diagnostic acumen.

ACUTE BOWEL OBSTRUCTION*

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THE outstanding consideration in the study of acute intestinal obstruction is the appalling mortality. Forty-one years ago, before the German Congress of Internists convened at Wiesbaden, Professor Curschmann deplored the bad results of surgical treatment and stated that medical measures gave fully as large a number of recoveries. The mortality with the conservative non-operative treatment in his clinic was 65 per cent. A year earlier at a meeting of the American Association of Physicians and Surgeons in this country, this subject was extensively discussed by Reginald Fitz and Nicholas Senn. It was brought out that the surgical mortality in the treatment of intestinal obstruction was 69 per cent. A large number of recognized authorities participated in the discussion and it was the consensus of opinion that after conservative measures had been tried for two days without success recourse should be had to surgery.

Today it is universally conceded that operation should be done for the relief of acute bowel obstruction as soon as the diagnosis can be made. Despite this general acknowledgment, cases come to operation almost as late as when it was uniformly agreed that conservative measures should be tried for two days.

Present day surgical results, though somewhat better than those obtained in the surgery of acute intestinal obstruction at the dawn of the era of abdominal surgery, exhibit no such striking improvement as can be boasted in the surgical treatment of most other acute abdominal catastrophes. Statistics from our municipal hospitals where cases of acute bowel obstruction are seen in large numbers indicate that the mortality is almost as great today as it was forty-two years ago.

C. Jeff Miller has recently published a study of the cases of acute intestinal obstruction admitted to the Charity Hospital and Touro Infirmary in New Orleans. In the five year period, immediately preceding his report, there were 343 cases with 207 deaths, a mortality of 60.9 per cent. In 1900, Gibson reviewed from various hospitals 1,000 cases of intestinal obstruction that had been subjected to surgery, with a mortality of 43.2 per cent. VanBeuren and Smith in 1925 published a collected report on 1,089 operations performed since 1900 with a mortality of 41.8 per cent.

Why do 40 to 60 per cent of patients operated on for acute intestinal obstruction die? Why does this high mortality still continue?

During the half century that has passed since the aggression of surgeons into the domain of the abdomen, strides of great consequence have been made in treatment; witness the improvement of results in the surgical treatment of acute appendicitis, acute perforations of the stomach and duodenum and other abdominal disasters of an acute nature. Today, death following an operation for acute appendicitis is synonymous with delay in the recognition of the ailment or procrastination in advising surgery. Similarly, death from perforations of duodenal or gastric ulcers signifies that an unreasonable length of time has intervened between the onset of the symptoms and the intervention of the surgeon. In 1922, Barber and Stewart reported twenty-four operations for perforated duodenal ulcer with two deaths; in the lethal cases surgical interference had been delayed for more than twenty-four hours. Today the mortality varies directly with the number of hours lost between the occurrence of the catastrophe and the closure of the perforation.

Why has not the remarkable improvement attending the surgery of other acute abdominal lesions extended to intestinal obstruction?

The results of a large number of experimental investigations have indicated that the absorption of a toxin producing profound and overwhelming effects on the organism is in large measure responsible for the mortality of the disease. Though recently several investigators have obtained results at variance with this explanation, many clinicians have been quick to seize upon the toxic factor, finding in this refuge some sol-

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ace for their misgivings and a satisfactory account for their poor results.

The fact remains, however, that all statistical studies indicate that there is a rising mortality in intestinal obstruction commensurate with delay in arriving at the diagnosis and procrastination in advising surgery. In cases operated upon at an early hour after the onset of symptoms, the mortality compares favorably with the surgery of other abdominal emergencies. In any number of papers on obstruction of the intestine, the difference in risk between those cases that come to operation early and those submitted to surgical intervention late is striking.

THE RISING MORTALITY OF DELAY (TUTTLE)

From onset of symptoms to operation	Total	Deaths	Per Cent
Under 6 hours	13	0	0.0
Under 12 hours	25	1	4.0
Under 24 hours	39	7	15.4
Under 48 hours	50	9	18.0
Under 3 days	70	17	24.3
Under 4 days	84	26	30.9
Under 6 days	99	37	37.4
All cases	128	53	41.3

In a consecutive series of 283 cases operated by Flint and reported by him in 1921, the mortality of operation for all cases of intestinal obstruction was only 15.6 per cent. Recently Holden has reported 135 cases of intestinal obstruction with a total mortality of 19.2 per cent. Of these 135 patients, 51 came directly to Holden. There were only three deaths in this group, a mortality of 6 per cent made possible by early recognition of the presence of obstruction and prompt surgical interference. The key to better results in the treatment of intestinal obstruction is early diagnosis.

PATHOLOGY

Classification:

From the point of view of a pathological division there are essentially two types of intestinal obstruction, viz., simple and strangulation obstruction. In the former group are included all cases in which a block in the continuity of the bowel exists; in the strangulation group are those which exhibit in addition to a mechanical block, evidence of deprivation of blood supply to the intestine.

The bowel wall above the obstructing mechanism is distended, hyperemic and thinned out in obstructions of an acute nature. Fluids and gas accumulate in the proximal segments in large amounts. Microscopically partial erosion of the intestinal villi or mucous membrane may be seen together with edema and hemorrhage throughout the layers of the bowel wall. The bowel distal to the obstruction is anatomically normal. The imprisoned loop in well-developed strangulation obstruction is infarcted, exhibiting discoloration and signs of tissue death. In simple obstruction to the continuity of the bowel, in which the dis-

CLASSIFICATION OF INTESTINAL OBSTRUCTION

A. *MECHANICAL*
- I. NARROWING OF LUMEN
  - 1. Strictures of bowel wall
    - a. Congenital { Atresia; Imperforate anus }
    - b. Acquired { Inflammatory; Traumatic; Vascular; Neoplastic }
  - 2. Obturation
  - 3. Compression from without (Especially pelvis and retroperitoneal duodenum)

  } SIMPLE

- II. ADHESIVE BANDS { Congenital; Inflammatory; Traumatic; Neoplastic } SIMPLE OR STRANGULATION
- III. HERNIA
  - 1. External—STRANGULATION (with only few exceptions)
  - 2. Internal—STRANGULATION (usually)
- IV. VOLVULUS—STRANGULATION
- V. INTUSSUSCEPTION—STRANGULATION

B. *NERVOUS*
- I. INHIBITION ILEUS (Paralytic) Adynamic—SIMPLE, OCCASIONALLY STRANGULATION
- II. SPASTIC ILEUS (Dynamic)—SIMPLE

C. *VASCULAR*
- I. THROMBOSIS AND EMBOLISM OF MESENTERIC VESSELS
- II. SEVERANCE OR INJURY OF MESENTERIC VESSELS (Operative or blunt trauma) STRANGULATION

tension is great enough to impede the free arterial inflow of blood to the intestine and egress of the venous return, the phenomena of devitalization as observed in strangulation obstruction may ensue. This occurrence is not infrequently observed in the cecum consequent upon the enormous distension of the colon occasionally accompanying carcinomatous obstruction in the sigmoid flexure. As a consequence, distension ulcers, perforation, and gangrene may follow, as are regularly observed in non-viable strangulated segments.

From the point of view of causative factors, three main types of intestinal obstruction may be recognized, viz., mechanical, nervous, and vascular. This division furnishes an adequate and satisfactory clinical classification, into one of which categories any observed variety of intestinal obstruction may be easily placed. By far the larger number of instances of obstruction are due to mechanical agents such as adhesive bands, hernia, neoplasms, intussusception, and volvulus.

Cause of death:

The discovery of Hartwell and Hoguet, that dogs administered saline subcutaneously may survive complete obstruction of the upper intestine for as long as three weeks, has lent considerable impetus to the observational study of bowel obstruction. Since these observations were made our ideas concerning the mode of death in obstruction have undergone considerable change. In this brief paper devoted primarily to the elaboration of clinical criteria on which an early diagnosis can be made, the pathological physiology of bowel obstruction can be dealt with in only a summarical fashion.

There are many features of the problem that still remain unsolved. Whether the intestinal content above the obstruction is more toxic than normal intestinal content is a debatable issue. Some experiments performed by Chunn and me indicate that it is not. Whether abnormal absorption occurs from the lumen of a distended bowel to which the blood supply is not compromised is an even more important issue. A few older well known experimenters (Enderlen and Hotz, and Clairmont and Ranzi) have shown that the obstructed bowel is less permeable to substances ordinarily absorbed than is the normal intestine; and Loucks and I were unable to obtain evidence of the absorption of histamine in simple obstruction when histamine was placed proximal to the obstruction. Though such abnormal absorption may obtain, I do not know of a single bit of evidence which indicates that in simple obstruction in which the bowel wall is viable that it does occur. There is, however, considerable negative evidence in which no proof of abnormal absorption has been obtained.

In strangulation obstruction, on the contrary, autolysis of the mucosa of the devitalized bowel quickly occurs and abnormal absorption through the mesenteric blood vessels occurs as well as via the serous surfaces of the peritoneal cavity when the strangulated segment is no longer viable.[4]

In experimental obstruction of the upper intestine in the dog, the subcutaneous administration of saline serves as an antidote not for abnormal toxins absorbed, but as a substitute for vital fluids lost by vomiting. When saline is not given, death usually occurs within 3 or 4 days and a marked increase in the blood nonprotein nitrogen, a decrease in the blood chlorides, and an increased combining power of the blood for carbon dioxide obtains (alkalosis). The administration of saline precludes the occurrence of these alterations in the blood chemistry and prolongs the life of the animal. Even a temporary administration of saline for a few days will often permit the animal to live just as long as though it were given each day.[24] Animals with a complete duodenal fistula die in a few days and exhibit the same blood chemistry changes as do dogs with obstruction at the same level.[22] The administration of saline prolongs their lives and obviates the occurrence of the blood chemistry changes.

Dogs with complete obstruction in the lower colon usually survive for comparatively long periods. In our laboratory one dog has survived such an obstruction for 50 days. The usual cause of death is dehydration and starvation. The administration of saline does not prolong their lives and blood chemistry alterations are usually not observed.

Many phases of the cause of death in acute bowel obstruction are still imperfectly understood, but it would appear that there is a fairly direct correlation between the function of the part of the intestinal tract concerned and the cause of death.[26] Obstruction of the upper intestine produces changes in the blood chemistry

and saline is an effective substitute. In low colon obstructions, alteration in the blood chemistry is unusual and saline exhibits no specific value as a remedial measure; similarly in experimental obstruction of the ileum. The dogs with low colon obstructions frequently survive for long periods whereas the dogs with ileal obstructions in which a good portion of the absorptive area is blocked out usually die within a week; saline does not prolong their lives. In strangulation obstructions, saline is of no special value as a therapeutic agent. These latter observations serve to emphasize the significance of the mechanical factors in bowel obstruction.

DIAGNOSIS OF INTESTINAL OBSTRUCTION

Symptoms:

Pain, vomiting, collapse, constipation, and meteorism have been the usual standards on which judgment has been based as to whether or not obstruction of the bowel exists. All of these phenomena may be observed at some time during the course of intestinal obstruction but their importance as measures of diagnostic value must be properly appraised. In his nonpareil text on the Principles and Practice of Medicine, the late Sir William Osler says, "The sequence of gastric, bilious, and finally stercoraceous vomiting is perhaps the most important diagnostic feature of acute obstruction." That this in part is true must be freely conceded, but it is decidedly unsafe to await confirmation of this succession of events. Similarly, absolute constipation, meteorism, and collapse may establish the diagnosis, but at a time when the patient is beyond hope and remedy.

Unlike other acute surgical lesions of the abdomen, simple obstruction of the intestine produces no local signs that help to corroborate the suspicion that obstruction may be present. Appendicitis, biliary colic, and perforation in the gastro-intestinal tract occasion definite physical findings that are of immense value in deciding the nature of the condition present. The recognition of the existence of acute inflammation of the vermiform appendix without the occurrence of local tenderness and rigidity would enhance the difficulties of diagnosis enormously; the number of cases coming to operation with peritonitis would be manifoldly increased should such difficulties exist.

Undoubtedly the absence of definite local physical findings more than any other factor accounts for the delay in the recognition of bowel obstruction. Were this point continuously borne in mind, the occurrence of crampy intermittent pain without abdominal tenderness would not be so lightly considered. At the same time, the absence of local symptoms should be of distinct value inasmuch as it obtains in no other serious surgical emergency that affects the abdominal viscera.

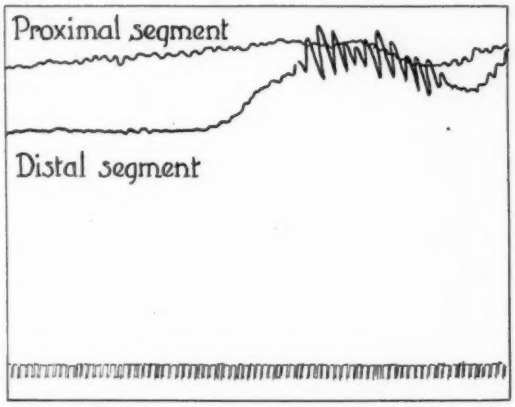

Fig. 1. Tracing made on a dog with complete intestinal obstruction of two days' duration. A balloon was inserted into the proximal segment and another into the segment distal to the obstruction. The peristaltic activity of this segment is apparent in the tracing.

Auscultation of the abdomen often lends the most valuable of all physical signs of obstruction. Visible peristalsis though regularly present in subacute and chronic obstructions is rarely observed in acute bowel obstruction. Loud peristaltic rushes like the bubbling sound produced by pouring water out of a bottle may be heard with the stethoscope when a mechanical obstruction exists. A dilated intestine in which considerable fluid and air are present is necessary to elicit these bubbling sounds. Near the point of intestinal occlusion these intestinal noises may attain an explosive force. Frequently a metallic tinkle may be heard, a sign of conclusive significance for the existence of a mechanical obstruction. As the fluid and air are carried downward toward the point of obstruction by peristaltic rushes, their progress is suddenly arrested and fluid and air which were intimately mixed in their advance tend to separate as the onward movement is halted. As the air rises to the sur-

face, a sound like drops of water falling in a rain barrel may be heard. The metallic character of the sound has a high pitch imparted by the tension of the dilated intestine. Such sounds may also frequently be heard over the abdomen after an abdominal operation when the paresis of the admission at the University Hospital; her physician, though suspecting bowel obstruction, had been unwilling to accept or commit himself to such a diagnosis because gas and feces continued to be expelled following the administration of enemas.

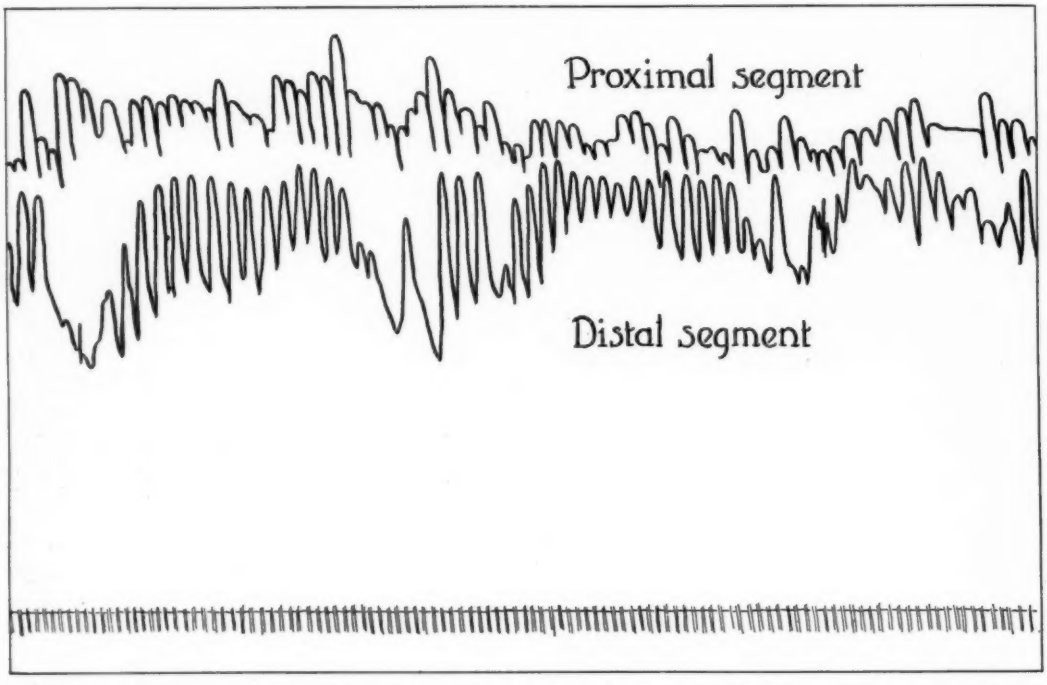

Fig. 2. A tracing made under conditions similar to those in Figure 1, with the exception that the record was obtained under spinal anesthesia. In this record the release from the inhibiting influence of the splanchnic nerves is apparent in the marked peristaltic activity of the intestinal contractions. The distal segment is fully as active as the proximal. The fallacy of considering the segment distal to the point of obstruction inactive is apparent from this tracing.

intestine incident to the operation begins to be recovered from.

Mistrust in enemas:

A diagnostic criterion upon which too much emphasis has been placed in the recognition of acute bowel obstruction is the inability of the patient to expel gas or feces following the administration of an enema. There exists a general tendency to disbelieve the presence of a complete obstruction as long as gas or feces continue to be returned with the enema. Recently a patient came under my observation with enormous abdominal distension and regurgitant feculent vomiting. She had been operated upon a few weeks previously, and had persistent intermittent colicky pain and vomiting for four days before

In the experimental laboratory it was found that, even after complete division and inversion of the ends of the intestine, enemas obtained gas as long as there was any present in the distal bowel.[25] By placing balloons in the bowel distal to the obstruction it was noted that the peristaltic activity compared favorably with that of the normal.[3] The bowel distal to the obstruction is physiologically as well as pathologically normal! Why shouldn't the bowel distal to the obstruction expel gas or feces following the administration of an enema? In high obstructions gas will continue to be expelled as long as rectal irrigations are given; in partial obstructions as long as some of the content of the bowel proximal to the obstruction finds its way into the distal segments gas will also continue to be ex-

pelled with enemas. Only in complete low obstructions are enemas of real value in the early recognition of acute bowel obstruction.

Value of x-ray in diagnosis:

It has long been recognized that the *x*-rays are of value in detecting bowel obstruction, but this agent has not been accorded the importance that it deserves in the diagnosis of acute intestinal obstruction.

When a single plate of the normal abdomen is made in the adult, gaseous shadows are usually observed in the stomach and colon. Gas is also present in the small intestine but the intimate admixture of gas and fluid in the small bowel precludes distinguishing the gas from its immediate surroundings. The demonstration of gas in the small intestine on the *x*-ray film is therefore decidedly abnormal and is indicative of the presence of a mechanism interfering with the normal rapid rate of transit through the small intestine. The ladder "patterns" described as the typical *x*-ray picture of bowel obstruction need not be awaited to make the diagnosis. Visible collections of gas in the small intestine of the adult are synonymous with intestinal stasis. In the experimental laboratory it has been found

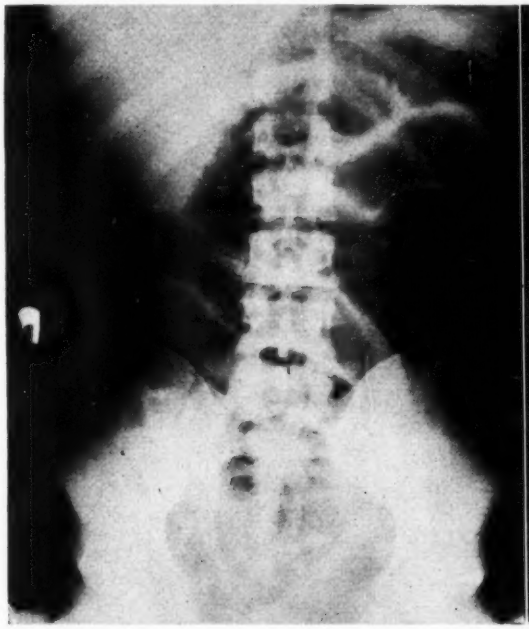

Fig. 3. A flat plate made of the abdomen in a case of peritonitis in which the entire intestinal tract is distended. In mechanical obstruction of the intestine the bowel above the point of obstruction alone is distended—another bit of evidence for the intestinal stasis being due to peristaltic ileus (inhibitive ileus) is that the intestinal wall between the dilated loops seems unusually thick in places; this appearance is afforded by the presence of exudate between the intestinal loops. This patient, of course, had abdominal tenderness and rigidity, which are almost invariably absent in simple intestinal obstruction. Auscultation of the abdomen failed to elicit loud peristaltic noises.

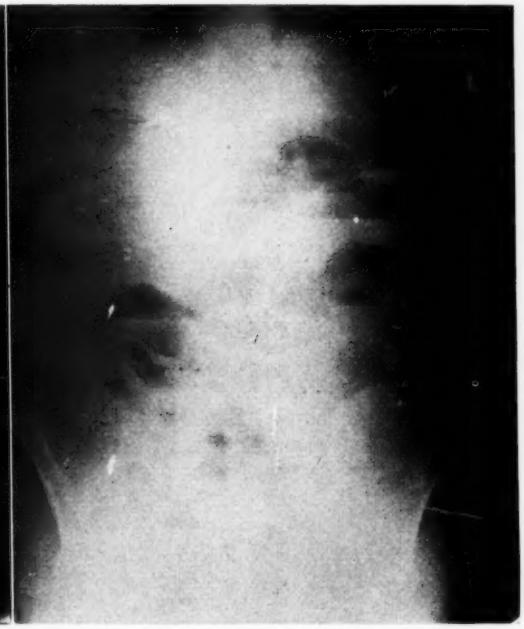

Fig. 4. Single plate of the abdomen made in the erect posture from the same patient as shown in Figure 3. The "fluid mirrors" in the small intestine indicative of intestinal stasis are well shown. The stethoscope in such cases serves as the best differential to determine whether the stasis is of mechanical or inhibitive origin.

that definite *x*-ray evidence by the single plate method may be obtained within four or five hours after the obstruction.[28] When the plate is taken in the erect posture "fluid mirrors" can be made out in which air is layered over the fluid. These afford concrete proof of the presence of intestinal stasis. In patients too ill to stand, the same information may be obtained by taking the plate with the patient on his side.

It is to be remembered, however, that the mechanism for the production of such intestinal stasis may be either mechanical or inhibitive. The stethoscope will serve to differentiate! In the inhibition or paralytic ileus of peritonitis, auscultation reveals a relatively silent abdomen, occasionally as silent as the "stillness of the grave." In mechanical obstruction on the contrary, loud

borborygmi and frequently metallic tinkles are to be heard.

The oral administration of barium is of no value in the recognition of most cases of acute bowel obstruction and usually only contributes to unnecessary procrastination in arriving at the diagnosis.

Fig. 5. A single plate taken of the abdomen of a patient who had complained of intermittent crampy pain of seven hour's duration. There had been considerable vomiting, but physical examination of the abdomen was negative. A previous enema had evacuated most of the gas in the colon; the loop on the left, however, indicates the abnormal presence of gas in the small intestine. Loud peristaltic borborygmi were heard on auscultation. The "ladder pattern" of intestinal obstruction as shown in Figure 3 is not necessary for the diagnosis of obstruction because the visualization of gas in the small intestine in the adult is significant of intestinal stasis.

Fig. 6. Plate made of the abdomen in the erect posture in a patient with intermittent crampy pain of twelve hours' duration. The "fluid mirrors" indicate the presence of intestinal stasis. The stethoscope as well as the crampy pains serve to designate the mechanical origin of the obstruction.

General condition:

Early in the course of simple obstruction the general condition of the patient is good. The crampy intermittent pain and nausea and vomiting make him ill at ease, but he may not appear especially sick. The body temperature is normal and continues so until late in the disease, when subnormal or, less often, elevations of slight degree may be recorded. There is ordinarily but slight quickening of the pulse and no change in blood pressure until prolonged continuance of the obstruction ushers in circulatory collapse as manifested by hurried feeble pulse, cold and livid extremities, and pinched and sunken features, an anxious expression and great restlessness. Leukocytosis is absent early; later, incident to the dehydration brought about by vomiting, high leukocyte counts may be obtained with similar increases in the hemoglobin and red blood cells. Blood urea elevation, plasma chloride decrease and an alkalosis occur with regularity in high obstructions only and are not an early sign of acute bowel obstruction. Late in the disease, the tongue is parched and great thirst is complained of; the urine is scant and of high specific gravity and may contain albumin and indican. Alterations in respiration are dependent largely upon the presence or absence of distension. Late in the course of intestinal obstruction rapid superficial respirations are the rule. The sensorium remains clear until near the end when the sinister warnings of euphoria, delirium and coma portend the approach of impending death.

One of the greatest evils in the management of cases of intestinal obstruction has been the administration of morphine under the impression that no serious disorder exists. Both physician and patient are pleasantly lulled into a sense of temporary security by this disguise, and the significant warning of pain is silenced until physician and patient are rudely awakened, but often

too late to undo the harm wrought by the irremediable delay. A patient presenting intermittent colicky abdominal distress without localizing findings should be watched carefully. Such a patient should be under careful hourly scrutiny and preferably in a hospital. If intermittent pain continues despite the return of gas following the administration of enemas, the abdomen should be opened. It is far better, as C. H. Mayo has so wisely said, to open an occasional abdomen unnecessarily than to await the textbook picture of intestinal obstruction. If the criteria of obstruction elaborated above are adjudged properly, such an occurrence will be most unusual.

SYMPTOMS OF STRANGULATION OBSTRUCTION

When in addition to a block in the continuity of the bowel, the intestine is deprived of its source of blood supply, a different clinical picture obtains than is observed in simple obstruction. Such a patient is more acutely ill; the pain is often agonizing in character; the pulse is usually quickened early in the course of the ailment and fever of 100°-101° is not unusual and higher temperatures may be recorded. The breathing is superficial and more frequent than normal. When a large segment of intestine is involved in the strangulating mechanism, symptoms of collapse may occur. Urinary suppression during the continuance of the strangulation is usual.

The clinical picture is determined by the degree of arrest of blood flow to and from the strangulated segment. When this is marked, symptoms of strangulation predominate; when the deprivation of blood flow is less prominent, the symptoms of mechanical obstruction dominate the picture. Every gradation from mimicry of the picture of simple obstruction to profound shock may be manifested.

In strangulation obstruction local physical findings in the abdomen are almost invariably present. Most strangulation obstructions present the features of a closed loop obstruction in which the distension of the obstructed segments progresses at a rapid rate. Consequent upon the infarction of the intestine, peritoneal irritation may usually be elicited by the demonstration of rebound tenderness. The accumulation of gas in the intestine and infarction of its wall may render the involved segment easily demonstrable by percussion and palpation of the abdomen. Wahl described this phenomenon a number of years ago and the presence of such a tympanitic mass is usually known as Wahl's sign. Such a loop may occasionally be noted on careful inspection of the abdomen. When a short segment of small intestine is concerned in the strangulation this sign may be absent due to the concealment of the involved intestine by other coils of bowel. Tenderness is usually present over the mass and

Fig. 7. Intestinal obstruction in a dog four hours after the establishment of complete obstruction in the lower ileum. Experimental study with *x*-ray of gaseous shadows in the intestine after obstruction indicates that reliable evidence can be obtained by this method within four to five hours after obstruction.

the latter imparts to the palpating hand an elastic resistance. The coil is usually fixed and exhibits no peristalsis. The intestine above the obstruction distends gradually but it has been found in the experimental laboratory that the gaseous shadows in the small intestine do not appear as early in strangulation obstruction as in simple obstruction and that the X-ray is therefore not as valuable in the early diagnosis.[10a] Loud, peristaltic rushes may be heard from the contractions of the intestine proximal to the obstruction. The bowel below the obstruction being normal may evacuate feces and gas on the administration of an enema. Later in the course of the ailment an inhibition ileus as may be seen in kidney colic or peritonitis may supervene and the abdomen becomes relatively silent and enemas are not expelled.

Vomiting of a reflex nature sets in early after

the onset of pain but ordinarily does not continue in the same degree as seen in simple obstruction. Blood in the stool is not infrequent and its occurrence in the vomitus in high strangulations has been observed. If the course of the disease continued uninterrupted, the beginning of the end of life is heralded for the sufferer by extreme restlessness, a feeble thready pulse, a cyanotic appearance of the skin which is cold and moist with sweat, and general signs of circulatory failure. If the strangulated bowel becomes non-viable, peritonitis supervenes and hiccough frequently manifests this occurrence. It is to be remembered, however, that, although the course of strangulation obstruction is ordinarily unusually rapid owing to the deprivation of blood supply of a portion of the intestine, such is not an invariable occurrence. When the strangulating features are less prominent, the progress of the disease simulates the picture seen in mechanical obstruction at the same level, and it has not been an extremely rare observation to find a short infarcted loop as a consequence of thrombosis of the vessels in a segment of the mesentery go entirely unsuspected for more than a week's time, manifesting some abdominal discomfort, but not sufficient to warn the surgeon or patient of the lurking danger.

TREATMENT

When to operate:

The essence of the treatment of acute bowel obstruction is early diagnosis and early surgical intervention. Operation must not be deferred until the nature and location of the obstruction are known. This information can be more accurately determined at operation with less risk to the patient than if surgical interference is put off until all these queries can be satisfactorily answered. When it is appreciated that it is most important to recognize bowel obstruction early a large number of operations will be in the nature of an exploratory procedure as to the type of lesion that will have to be dealt with. Unfortunately, at the present time a large proportion of the operations performed for the relief of bowel obstruction are in the nature of resuscitations, and failures and bitter disappointments are numerous.

Preoperative preparation:

When the decision to operate has been made, no great time should be lost in preoperative preparation. During the hour that preparations are being made in the operating room, the patient's stomach should be evacuated with a tube and a liter or two of normal saline given intravenously and subcutaneously. There is an impression not infrequently encountered amongst physicians that it is more important to put off operation for eight to ten hours, during which time the patient's condition is being "built-up" with free administration of glucose and saline. Though salt and water are important agents in combating the dehydration of obstruction, it must be remembered as has already been pointed out above that only in high obstructions do these therapeutic agents exhibit the virtues of a specific.

How to operate:

When patients come to operation early the choice of an anesthetic is not so important. But to administer inhalation anesthesia to a patient with regurgitant vomiting and considerable distension, even though the stomach is emptied by a tube immediately before the administration of the anesthetic, is a hazardous undertaking.

Spinal anesthesia is the ideal analgesic agent for cases of intestinal obstruction. At the University Hospital we have employed it with complete satisfaction to the exclusion of all other types of anesthetics in intestinal obstruction except in infants, in whom local infiltration of the abdominal wall with procaine combined occasionally with light ether anesthesia usually suffices. It is somewhat outside the province of this paper to discuss the stimulating effect of spinal anesthesia on the distended bowel in paralytic ileus. No harm is to be anticipated, however, in administering spinal anesthesia because of the induced increased intestinal activity in the presence of a mechanical obstruction when immediate operation is to be performed. The early history of the employment of spinal anesthesia is a dark chapter, but recognition of the fact that the Trendelenburg position and not the upright posture protects the patient, and the introduction of ephedrin to support the blood pressure have made its use safe. Within the past year I have been enabled with its employment to operate successfully upon several late cases that would not have tolerated operation under inhalation

anesthesia. Only in moribund patients is its use contraindicated.

The incision:

Unless it is definitely known just where the obstruction is, it is best to make a mid-line subumbilical incision, or a midright rectus opening of the peritoneal cavity. Most instances of small bowel obstruction can be approached satisfactorily through such an opening. In the presence of a large bowel malignancy in which a colostomy is found to be the procedure of choice, a short oblique inguinal incision can be made in either groin and the establishment of the colon decompression facilitated through the midline incision.

Location of obstruction:

In locating the point of obstruction, it is always wisest to hold up the abdominal wall first with retractors and look inside. The employment of spinal anesthesia permits easy and wide retraction of the abdominal walls and frequently exposes the site of obstruction, especially if the intestine is adherent to the parietal peritoneum. In disregarding this simplest of procedures the obstruction may not be located after diligent search with the hand in the abdomen. Short of a routine "going over" of the entire intestine, it is the most efficient method by which to locate the obstruction.

Many years ago Greig-Smith pointed out that a distended coil in the proximity of the site of occlusion will usually present itself into the wound. If the point of obstruction does not immediately come into direct vision, pursuit of the coil in the direction that presents itself least readily will usually lead to the obstruction. In addition, the intestine immediately above the point of obstruction will be hyperemic as well as dilated. When cases of intestinal obstruction are operated upon earlier, exploratory examination can be more deliberate and these signs are not as important. At least some of these cases, however, will always be with us and we must be prepared to treat them most efficiently.

Choice of procedure:

The most important decision the surgeon has to make when operating for intestinal obstruction is whether it will be possible to do the operation of election, viz., to find and relieve the obstruction and restore the continuity of the bowel or whether he must content himself with a simple drainage operation. The nature of the obstruction will in part decide this matter. In a strangulation type of obstruction in which a segment is deprived of its blood supply it is apparent that a choice of the magnitude of operation that the patient can best tolerate must not determine the procedure to be followed. Relief of the mechanical block in the bowel by enterostomy or release of the strangulating mechanism alone will not suffice; excision or exteriorization of the devitalized bowel must also be done. The continuance of such a segment in the abdomen is not compatible with recovery. The performance of a simple drainage operation in the presence of strangulation obstruction advanced to the degree of loss of vitality is therefore futile.

Immediate determination as to type of obstruction present must, therefore, be decided. The occurrence of bloody fluid in the peritoneal cavity signals the presence of a strangulation obstruction. In carcinosis of the peritoneum or in a low grade peritonitis, fluid may be present, but it is usually serous in character. An infarcted coil of intestine lends confirmation of the presence of a strangulation obstruction.

The course of action must depend solely on whether or not the bowel is viable. The return of normal color to the bowel and the return of pulsation in the vessels of the mesentery after reduction of the strangulation herald the viability of the bowel. In doubtful cases the decision as to what is best to do may be a difficult one. When the condition of the patient permits, a resection with end-to-end or lateral anastomosis is the most conservative procedure; damaged bowel proximal to the strangulation must be excised generously. It is a hazardous procedure to perform an anastomosis on a distended hemorrhagic bowel. In ill patients it is much the wiser plan to exteriorize the gangrenous bowel even in the small intestine and perform a secondary anastomosis later. If the fistula following exteriorization proves to be high in the small intestine the content from the upper loop should be collected with a catheter and put into the distal segment. The bowel wall of the strangulated segment may be edematous, but there should be no sharp line of demarcation of color between the normal and the involved intestine. When edema persists, the normal luster and

sheen of the peritoneal surface of the bowel may not return promptly. Normal contractile response to pinching is absent in the nonviable bowel.

When a strangulation obstruction is not present, the surgeon in his selection of operative procedure should permit himself to be guided entirely by the patient's condition. In the early case, the operation of election may be performed, viz., the determination of the location of the obstruction and its release. In the precarious risk, the absence of a strangulation obstruction is indication for the performance of decompression by enterostomy. The determination of the location and exact nature of the obstruction in such cases is made at the risk of inviting disaster. Suppression of curiosity on the part of the surgeon at such a time will afford him an opportunity to determine later in a living patient what need be done. It should be remembered that, though the persistent existence of adhesive bands in the small intestine are potential forerunners of subsequent intestinal obstruction, such a patient may find himself so free from symptoms as to be unwilling later to submit to more surgery.

In early cases of simple obstruction, location of the point of obstruction and evisceration of the intestine in its search may be practised with safety and equanimity. Evacuation of the intestinal content by enterostomy in such cases may be dispensed with as unnecessary. All anastomotic procedures on the dilated intestine are to be religiously avoided. Tissues partially devascularized by long continued compression of the blood vessels of the bowel wall consequent upon the intestinal distension do not permit safe suture.

In postoperative obstructions, enterostomy as a single therapeutic agent serves its widest field of usefulness. Consequent upon the subsidence of intestinal distension brought about by enterostomy, the continuity of the intestine is frequently automotically reestablished and the enterostomy opening closes spontaneously when the safety vent has served its purpose upon withdrawal of the tube.

The statistical mortality in this group of cases is surprisingly high. The late recognition of the cases is in large part responsible for this high death rate, but the tendency to performance of operations of election rather than the adaptation of a procedure suitable to the precarious condition of the patient contributes in no small degree to the persistence of a forbidding mortality in this group. Evisceration of the intestine to locate the site of obstruction, or the manual manipulation and examination of long segments within the abdomen, and prolonged operative procedure incident to their performance robs the ill patient with late simple obstruction of a large margin of his chance for recovery.

Selection of site for enterostomy:

A distended coil of intestine above but near the point of obstruction should be chosen as the site of enterostomy. The deliberate performance of a jejunostomy in each case is not only unnecessary but harmful. The exponents (Bonney) of high drainage of the intestine in obstruction have insisted that the tapping of the fluid and gas containing segment is all-important. In addition, it was erroneously contended that such a vent permits escape of the most toxic intestinal content. The danger of a high intestinal fistula, per se, has been adequately demonstrated. Haden and Orr have been able to show that in the dog even partial escape of the jejunal content through catheter drainage always proves fatal. High jejunostomy therefore unless inevitable because of the location of the obstruction is to be deliberately avoided.

Technic of enterostomy:

In the performance of enterostomy fine needles and suture material, preferably silk, are to be employed. When conditions permit, it is a good plan to draw out the loop upon which enterostomy is to be done and isolate it in one or be tween two rubber covered clamps. Puncture of the bowel wall with a fine needle followed by withdrawal of the content of the loop with an aspirating syringe will obviate the occurrence of leakage from the stitch holes. A urethral catheter (No. 16 French) is then buried in the bowel wall by the Witzel technic. When possible the catheter should be drawn through the omentum and the latter should be fixed to the anterior abdominal wall. By placing the catheter on the stretch and making a superficial circular cut around it, about 4 or 5 inches from the eye, a groove is secured in which a fixation suture may be placed. When the traction on the catheter is released, the groove and knot in the suture are scarcely apparent. A fine chromic catgut suture

is best employed to secure the catheter to the bowel wall. This refinement obviates the catheter's coming away before it has wholly fulfilled its intended purpose.

I do not believe that the employment of the Moynihan evacuation tube has any advantage; it exposes the intestine to unnecessary trauma and precious moments are wasted. When the distension is extreme, immediate evacuation of the bowel on the operating table by a trocar before suturing the catheter in place is commendable.

Postoperative treatment:

After operation the patient should be placed in bed so that respiration is unhampered. Semi-Fowler's position usually gives most comfort Physiologic sodium chloride solution is to be given daily subcutaneously with daily intravenous glucose (10 per cent) administration until the patient takes fluid well by mouth. The intake and output are to be carefully recorded; the vomitus or gastric retention recovered by expression are to be subtracted from the fluid intake. An optimum intake for a patient with intestinal obstruction in whom a considerable amount of fluid is being continuously dumped into the intestine, is in excess of the requirements of the ordinary abdominal surgical patient. In intestinal obstruction, only when salt begins to be excreted by the kidney does a diuresis occur. Three to four thousand cubic centimeters of fluid may well be utilized for the first few days. When the urine output approximates the normal, the intake may be decreased.

The patient with an enterostomy high in the intestine must be carefully watched for evidence of ill consequences from the loss of the high intestinal juices. When the effects of the obstruction have been successfully combated, the catheter may be intermittently clamped to determine the patency of the intestinal channel below. In the event that the enterostomy presents the only avenue of escape for the intestinal secretions, another catheter must be placed in the distal bowel into which the fluid collected from the oral loop may be placed, or a more direct attack on the obstructing mechanism must be made. The optimum time for such a procedure is when the patient exhibits the greatest improvement from the intestinal decompression.

SUMMARY AND CONCLUSIONS

The establishment of true criteria of bowel obstruction early in its course will help more than any single factor in bringing patients to operation at an advantageous hour and to a definite lowering of the present appalling mortality of the disease. Our textbook descriptions of intestinal obstruction should be labelled Antemortem Signs of the Disease. They do not indicate interference with the continuity of the intestinal lumen but herald impending dissolution. Regurgitant vomiting and distension must cease to be criteria awaited before a diagnosis can be made. Collapse in simple bowel obstruction should be relegated to the delineation of that antemortem status in which all vital functions rapidly depreciate and from which escape or resuscitation is unlikely.

Intermittent crampy pain, accompanied by nausea and considerable vomiting without abdominal rigidity or tenderness, is strongly suggestive of an acute block in the continuity of the intestine. The detection of loud intestinal noises on auscultation of the abdomen and the *x*-ray visualization of gas in the small intestine lend sufficient proof to make the diagnosis. The continuance of colicky pain despite the return of gas and feces on the administration of enemas constitutes sufficient indication for immediate operative intervention.

The management of cases of bowel obstruction in accordance with the principles outlined here will lead to the striking improvement in surgical results long since obtained in the surgery of other acute abdominal disasters.

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HALEY'S M-O MAGNESIA-OIL NOT ACCEPTABLE FOR N. N. R. AND MAGNESIA-MINERAL OIL (25) HALEY OMITTED FROM N. R. R.

The Council on Pharmacy and Chemistry reports that Haley's M-O Magnesia-Oil (exploited with the emphasis on "Haley's M-O") is the name under which the Haley M-O Co., Inc., has marketed a mixture of magnesia magma (milk of magnesia) and liquid petrolatum; that the firm requested acceptance of the product for New and Non-official Remedies stating that it is composed of magma magnesiæ 75 per cent by volume and liquid petrolatum 25 per cent by volume, that the Council refused admission of the product to New and Non-official Remedies because the use of a mixture of liquid petrolatum and magnesia magma in fixed proportions under an uninforming name is detrimental to rational prescribing; and that the preparation was accepted after the firm had adopted the name Magnesia-Mineral Oil (25) Haley and had revised its advertising to make it acceptable. After the Council had repeatedly been obliged to object to the advertising, the firm wrote to the Council that its attempts to meet the requirements of the Council were proving financially unprofitable and that it had decided to go back to the old name, "Haley's M-O" This means that physicians will again be asked to use this simple pharmaceutical mixture under an uninforming name. The Council directed the omission of Magnesia-Mineral Oil (25) Haley from New and Non-official Remedies and declared Haley's M-O Magnesia-Oil ("Haley's M-O") unacceptable for New and Non-official Remedies. (Jour. A. M. A., April 5, 1930, p. 1067.)

FRACTURES OF THE PELVIS*

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THE occurrence of fracture of the bones in man goes back to the dawn of history; Cotton says—in speaking of ankle joint fractures—since the Nordic first wore brogans. The study of Egyptologists proves that many thousands of years ago bones were broken and tied up with splints much as is done today.

We are living in the so-called "machine age," and while the industries have surrounded the machine with every possible safety device and have trained their employees to follow the slogan "safety first," still, because of the great expansion in industrialism, trauma still causes many injuries and the surgery of trauma is receiving more attention than ever before in the history of medicine. Interest in traumatic surgery and fractures particularly has been stimulated by the compensation laws, by the greater demand that has been made on the surgeon for good results, by compensation boards, by insurance companies, by employers, by the injured themselves, by the urge of our economic system to cut down costs and return the injured to work as soon as possible and by the increasing knowledge that has come from the use of the *x*-ray.

In reviewing the last 683 consecutive fractures occurring from 1922 to 1930 in our work at the Shipman Hospital, in which the service is principally with the iron mining industry, we have had nineteen pelvic fractures. From these figures it was found that pelvic fractures have occurred in about 3 per cent of all fractures. This is about the percentage reported at the present time by others. The consensus of opinion now seems to be that fractures of the pelvis are occurring much more frequently in proportion to the total number of fractures than they did thirty years ago and we find this true in our work. In a series of 324 fractures occurring in our work before 1910 only two pelvic fractures are recorded, whereas in this present series of only a little over two times as many fractures we have had nine times as many pelvic fractures. Sir William Wheeler of Dublin states that the figures of the older authorities as Von Bergmann, Mikulicz and others indicate that fractures of the pelvis constitute only 0.3 per cent of the total number of fractures. He feels that this is much lower than our present rate.

The cause of the decided increase in the number of pelvic fractures as compared to former times, and also in proportion to the total number of other fractures, is doubtless our "machine age" mentioned above; that is, the great development of machinery in industrialism, the great expansion of industry and also the great development in rapid transportation and in this connection especially the general use of the automobile by the public. The aeroplane can be included in this rapid transportation group. Since the advent of the automobile, more women and children have suffered pelvic fractures in proportion to the total number of persons than formerly. This of course is due to the fact that formerly nearly all pelvic fractures occurred in industry and therefore mostly amongst men. Now many women and children as well as men ride in automobiles and also ride in other vehicles of rapid transportation and receive pelvic fractures in accidents of transportation and also on the streets, as pedestrians, by being struck by these rapid transports. We must not forget, however, that the industrial organizations are doing everything in their power to cut down the total number of injuries by placing all safeguards possible around all machinery to prevent men from being injured and also by training the man himself in safety-first methods so that he will be careful of his own safety; and transportation companies are doing likewise. But, we can not say as much for the people themselves who in driving their own automobiles do not show, in the mass, the same degree of care for prevention of accidents that the industries do. Automobile accidents are increasing, and whereas in former times pelvic fractures were cared for in hospitals connected with industry, now, with the automobile in such general use, any physician anywhere may be called upon to treat a severe pelvic fracture any time.

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It is interesting to note that just as the seriousness of a head injury depends not on the bony injury or fracturing of the skull but on the injury of the contents of the skull, namely, the brain and its coverings, so the bony injury or fracturing of the pelvis is not nearly so serious a matter as the injury that may have occurred to the soft structures within and about the pelvis. Thus a torn urethra, a ruptured bladder, a ruptured kidney, a perforated intestine, a ruptured blood vessel of which the iliacs, the obturators, gluteals, and pudic are most likely to be injured, is far more serious than multiple fractures of the pelvis and separation of the symphysis pubis or sacro-iliac joints.

It is rather remarkable the large amount of fracturing and displacement of fragments that may occur in the pelvis in which an excellent functional result is obtained in spite of a poor anatomical result. Perhaps this is more true of the pelvis than of the fracture of any other bones. Nevertheless, we should strive to obtain as good anatomical reposition as possible, just as we do in all other fractures. Striving to get perfect anatomical reposition has become almost our greatest thought in treating fractures since the use of the *x*-ray. Patients and their friends seem to expect perfect end-to-end "cabinet joiners" apposition when shown an *x*-ray plate of a fracture reduction; and likewise courts and juries. This consequently has a tendency to force more attention to perfect anatomical reposition than to perfect function—perfect function, however, being the far more important consideration. Nevertheless, we may say in general in the handling of fractures, the better the anatomical result, the better the functional result. Yet good functional result may be obtained without end-to-end apposition, especially in shaft fractures, if good alignment is maintained. In fractures near joints and into joints we like to get as perfect apposition as possible.

In this series of nineteen pelvic fractures, there were two deaths (a little over 10 per cent). Other reports show a similar mortality rate. Noland and Conwell in reporting 125 pelvic fractures had twenty deaths (16 per cent). The mortality rate shows the seriousness of many of these pelvic fractures. There were seventeen males and two females. All were adults, ages ranging from nineteen to sixty-six or an average of 37.5 years. The total number of hospital days was 736 or an average of almost thirty-nine days in the hospital in the whole nineteen cases. Counting the twelve persons who actually stayed in the hospital for the full time of hospital treatment the average time was fifty-seven days, the longest 128 days and the shortest twenty-two days. Noland and Conwell in their series of 125 cases report an average of sixty-five days in the hospital, the longest period being 133 days. The average time before resuming work of twelve patients traced was 194 days. The longest time off was 437 days. The shortest was ninety-three days. In Noland and Conwell's 125 cases, the average number of days lost was 175. The total number of pelvic fractures was forty. The total number of other fractures accompanying the pelvic injury was sixteen. The sum total of all fractures was fifty-six. The largest number of pelvic fractures in one person was seven. The largest number of other fractures in one person was seven. The largest number of all fractures in one person was nine.

Classified anatomically there were the following fractures:

Ilium	10
Pubis	
Horizontal or superior ramus	10
Descending ramus	8
Body	2
Total pubic bone fractures	20
Ischium	
Ascending ramus	2
Sacrum	1
Acetabulum	1
Symphysis pubis separation	2
Sacro-iliac separation	4

The complications occurring were: ruptured kidney, injured ureter, ruptured bladder, traumatic asphyxia, fractured ribs, fractured transverse processes, spinus processes and lamina of the lumbar spine, fractured scapula, double fracture of the clavicle, spinal cord injury with paralysis, foot drop due to peroneal nerve injury, hematomas at various regions of the pelvis and back, contused ankle, scalp wound, concussion of the brain, and blood in the urine. G. G. Davis regards peroneal nerve injury as occurring originally in the lumbo-sacral cord at the pelvic brim.

The causes of injury were as follows: eight occurred in the iron mining industry due mostly to being squeezed or rolled between a car and a

timber; four were from automobile accidents, three while riding in cars and one by being struck by a car; two from gunshot wounds, accidental; one by being struck by a large piece of steel in construction work; one falling from a height of thirty-five feet; one by being knocked down by a cow and jumped on; and two in the lumbering industry, one from a falling tree and one from falling down a skidway and being struck by rolling logs. Eleven were industrial and eight civilian accidents. Blood in the urine occurred five times. Noland and Conwell reporting 125 pelvic fractures noted blood in the urine in sixty cases.

Many of these patients with pelvic fractures are in a severe state of shock on admission and first need to receive treatment for this condition. Their greatest complaint is of pain. It has been stated that some of them fear they will "fall to pieces." In the severe cases the abdomen seems continuous with the thighs. The Poupart ligament demarkations appear obliterated. There will often be other injuries besides the pelvic fracture, as the injuries are usually produced by great violence, although some are produced by slight trauma and have no complications or shock and no displacement of fragments and are consequently very simple to treat. An *x*-ray should be taken as soon as possible, and since the advent of the Bucky diaphragm this has become comparatively easy. It will immediately show the bony injury and obviate the necessity of too severe a physical examination in a very seriously injured patient. It will not, however, tell us the amount of injury to the soft parts, which, after all, is the most important thing to know. A specimen of urine should be obtained at once, by catheterization if necessary. If gross blood is found, an immediate attempt should be made to find its source. It may of course come from any point of the genito-urinary tract. Blood, however, may occur in the urine without any rupture of a viscus, probably due to a traumatization of the mucous membrane along the tract. Presence of blood indicates injury but gives little help in determining the source. If the urethra is ruptured, the catheter usually meets obstruction near its membranous portion. A perineal urethrotomy should be done if the urethra is seriously injured. If the ends of the urethra can not be found or united, the bladder should be opened suprapubically and a retrograde catherization done, another catheter being passed from the distal end of the urethra. The ends of the two catheters are threaded together, and traction on one directs the other through the two portions of the urethra. The ends of the urethra can thus usually be sutured over the remaining indwelling catheter. If the ends can not be brought together, they are simply held with tension-sutures. The catheter is left in place and the urethra will usually fill in the gap. This is a similar method to the one McArthur uses in repairing a cut common duct. It is surprising how well the urethra will take care of itself if given a chance. For a number of months after healing is secured, it will be necessary to pass sounds to prevent strictures. We have used this method in repairing injured urethras although we have had no injured urethras in our pelvic fractures.

Peacock in an analysis of twenty-eight cases of ruptured urethras found 71 per cent associated with fractured pelvis. On the other hand, he found only 9.8 per cent of fractured pelves that presented rupture of the urethra.

It is not always easy to decide whether a patient has a ruptured bladder and often requires fine judgment in making a diagnosis. The diagnosis may often be made only at operation, since the evidence will usually point to the probable rupture or perforation of some viscus in the abdomen and the condition of the patient usually does not warrant prolonged study. On opening the peritoneum, blood and urine are found and a tear in the bladder exposed.

Livingston, in the American Journal of Surgery, states that bladder rupture is by no means frequent (being encountered in one in 3,000 general surgical cases) but the high mortality of this accident justifies its careful study (30 to 80 per cent mortality even under the most favorable conditions). Bladder rupture is always due to trauma but the accident is favored by vesical distention, alcoholism and pre-existing diseases of the vesical walls. It is often observed with fractures of the pelvis. Twenty-five per cent of the patients with pelvic fractures suffer simultaneous bladder injuries. Ninety per cent of cases of bladder rupture occur in males due to the more violent nature of their activities, also to the greater depth of the female pelvis and the interposition of the uterus between the bladder and the vertebral column.

Quain, in 1916, reviewed the literature and reported 126 cases of rupture of the bladder all associated with fracture of the pelvis. The first case reported occurred in 1795. The patient, a man, was crushed under a wall and as a result there was a rupture of the bladder and the symphysis pubis was separated. The patient died 56 hours later. Autopsy showed infiltration of urine and peritonitis.

Fig. 1. Case 4.—J. V. Fracture of the left ala of the ilium; fracture of the horizontal ramus of the left pubis; fracture of the horizontal ramus of the right pubis; fracture of the descending ramus of the right pubis; fracture of the transverse processes of the two lower lumbar vertebræ. Ruptured bladder—death.

Fig. 2. Case 6.—E. M. Fracture of the horizontal ramus of the right pubic bone; fracture of the descending ramus of the right pubic bone; fracture of the horizontal ramus of the left pubic bone; fracture of the descending ramus of the left pubic bone. Later complicated by an attack of acute appendicitis—operation—recovery.

When bladder injury is suspected and the patient has a strong desire to urinate but is unable to do so, immediate catheterization should be done. If no urine returns, it may be due to an anuria from shock or to an intraperitoneal rupture with escape of urine into the peritoneal cavity. Usually the catheter passes freely through the urethra but only a small amount of urine is obtained with some blood, or more bloody urine may be drained away than could have been contained in the bladder, the urine having poured back into the torn bladder from the peritoneal cavity. In making a diagnosis some authorities suggest that a measured quantity of fluid should be injected and the fluid returned carefully noted and checked. There are disadvantages to this procedure. If the injected fluid leaks through a rupture into the peritoneal cavity, the desired information is obtained but the shock from which the patient suffers becomes more pronounced. If it returns in full quantity, it may have passed through an extraperitoneal rupture into the space of Retzius and returned with no appreciable diminution of quantity. Under such circumstances, the surgeon is deceived. In bladder ruptures either extra- or intraperitoneal, if the rupture is very small, a considerable quantity of urine, probably blood stained, will be recovered by catheter, as very little may leak out and no information as to the presence or absence of the rupture will be definite. Cystoscopic examination will assist in these cases. Injection of 150 to 200 c.c. of air into the bladder, followed by an *x*-ray, has been advocated. If air has passed into the peritoneal cavity it will form a bubble in the subphrenic area and if not visualized an intact bladder is presumed.

Rupture of the bladder is often produced by a spicule of bone, perhaps more often than by the bursting effect of the trauma. This was true in Quain's report of 126 cases. The rupture is either intra- or extraperitoneal. If intraperitoneal, which is more common, it is usually near the fundus or posteriorly. The urine escapes either into the space of Retzius and percolates toward the anterior wall or, as is more often the

case, it escapes into the peritoneal cavity. In operation, the abdominal cavity is opened, all urine and blood removed from the cavity, the bladder sutured tightly with catgut (silk has also been used), and a self-retaining catheter is left in the bladder. Sometimes it is necessary to drain the abdominal wall down to the suture line of the bladder. Suture of the bladder has been considered unnecessary by some authorities if the bladder is thoroughly drained. We would ordinarily prefer suture. If the tear is extraperitoneal, it is usually on the anterior surface, either in an accessible portion or down below in the neighborhood of the trigone and post-prostatic area, and if not drained by immediate operation urine will eventually come to the surface through the inguinal canal, the femoral opening into the perineum or prevesical space. In our series there was only one case of proven rupture of the bladder.

Fig. 3. Case 7.—A. L. Fracture of the right crest of the ilium; fracture of the twelfth rib on the right side with injury to the right kidney and ureter.

Fig. 4. Case 9.—A. S. Compound fracture of the right crest of the ilium with displacement; separation of the symphysis pubis; separation of right sacro-iliac joint. Blood in the urine.

In only one of our cases did there appear to be injury to the kidney. We thought the patient probably had a rupture of the kidney. This diagnosis was based on exclusion of the urethra and bladder, the presence of nearly pure blood in the urine, the large mass that formed in the region of the kidney which seemed to be due to hemorrhage, the history of the injury (the patient being struck in the region of the kidney by a heavy piece of steel), and the *x*-ray findings of fracture of the twelfth rib and of the ilium on the same side. We did not operate because, after treatment of the shock, the patient within a few hours began to improve and at no time did he show signs of infection or extravasation. No cystoscopic examination was made, but later a functionless kidney was found by the urologist, absolutely no urine coming from the ureter, the ureter being closed at its junction to the pelvis of the kidney. It was therefore assumed that the ureter was injured at the time and in healing had closed completely and thus an auto-nephrectomy had occurred, the result a functionless kidney.

The principal symptoms of rupture of the kidney are hematuria, pain and a mass in the region of the kidney. The mass is usually due to the bleeding from the kidney. The rapidity with which it increases in size gives a rough estimate of the severity of the kidney hemorrhage. When the kidney is injured, such as in contusion or rupture, the question arises as to what other organs have been injured, if any, and whether or not the kidney injury demands operative treatment. After ruling out injury to the urethra and bladder, the two outstanding indications for operation are hemorrhage and infection. In the period immediately following the injury, one seeks, especially, any evidence pointing to severe

and continuing hemorrhage. Repeated examinations of the blood pressure, hemoglobin and counts of red and white cells of the blood are of assistance in determining the continuance of hemorrhage, as stated in Dean Lewis's Surgery. Drainage for infection of hematoma or extravasation of urine into the lumbar gutter, and suture of the kidney or nephrectomy for control of hemorrhage, are the usual operative procedures when operation is decided upon.

Fig. 5. Case 14.—M. B. Gunshot fracture of the pelvis: Fracture of the left ilium—injury to sacro-iliac joint; fracture of two transverse processes of the lumbar vertebræ.

Fig. 6. Case 15.—G. J. Separation of the symphysis pubis; separation of the right sacro-iliac joint. Traumatic asphyxia.

Bellevue Hospital, New York City, reports from 1914 to 1929 fifty-two cases of kidney injury, complicated in only one instance by pelvic fracture. Of these fifty-two cases, twenty-eight were simple contusions; six of these were operated on and all got well. There were eighteen cases designated as laceration or rupture of the kidney. The mortality record shows the seriousness of kidney injury as a class. Eighteen of the fifty-two patients (34.6 per cent) died; thirteen died without operation; five died following operation. Of the eleven cases operated on for contusion or rupture, six lived. Nephrectomy was done five times. There is probably no fixed rule to follow in these cases. Each case must be treated on its merits. The surgeon should stand ready to do an exploratory operation in the presence of alarming or continued hemorrhage in an effort to control it. Kidney suture or nephrectomy may be required.

Following the treatment of the complications of pelvic fractures, the patient should be placed in bed and treatment of the fracture itself begun. In most of our cases, this was done by simple rest in bed flat on the back, using sandbags for support to the legs and pelvis. In some of the cases, we have used, in addition, traction with abduction, especially if there was upward displacement; and an encircling belt which could be tightened gradually, especially in sacro-iliac and symphysis pubis separations. Adhesive strapping was also employed.

We have used the plaster cast in only one case, in which the acetabulum was fractured, and in this case traction was employed previous to the application of the cast. This had to be removed as it was not satisfactory and traction again resorted to with suspension of the leg. We did not operate on any of our cases. In one case in which there was bad displacement of the fragment of the horizontal ramus of the pubis we felt that a better anatomical result might have been obtained by operative replacement, but functionally the result is good. At present we are using a fracture bed with overhead frame which allows extension in all directions and also overhead suspension. It has also a trundle-bed arrangement for raising and lowering the patient for use of a bed-pan, caring for the patient's back and other necessary nursing care without

necessitating turning or lifting or disturbing the patient in any way or moving the extension or fractured parts, the patient being suspended by canvas cross pieces. We feel that this is very valuable in the treatment of pelvic fractures, fractures of the spine, fractures of the neck of the femur and all other femur fractures. Of course, the bed can be used in any fracture that requires extension and rest in bed.

Noland and Conwell, who recently reported 125 pelvic fractures in their work with the Tennessee Coal, Iron and Railroad Company, treat their patients by an overhead pelvic suspension frame. Suspension is obtained by a canvas sling or hammock which extends from the upper third of the thighs to the lower dorsal region. We quote from them as follows:

"This hammock acts as an immobilizing factor because of the lateral compressive action on the pelvic girdle, the force being approximately equal to the body weight. This force is continually in action and as relaxation of the muscles take place there is a constant disposition of the displaced fragments to fall into their natural position. As a general thing, marked relief of pain is noticeable within the first few hours following the application of the pelvic hammock.

"Suspension is supplemented by traction on the legs and thighs with about fifteen to twenty degrees of abduction at the hip. This relieves the marked spasm of the strong adductors, prevents scissoring of the thighs, tends to pull the fragments back into place and prevents the limited abduction so commonly observed following these injuries. Sufficient weight is used on the hammock to counterbalance the weight of the patient but not enough to raise the patient completely from the bed. Weights used for leg traction vary from four to eight pounds. A spreader is used in certain cases to prevent too great a lateral pressure by the hammock. Lateral pressure of the hammock may at once cause an overlapping at the point of the fracture unless it is offset by the intelligent use of the spreader. The degree of lateral pressure can easily be governed by the lowering or raising of the wooden spreader. The spreader should be the exact width of the individual's pelvis within the canvas sling. The use of the spreader should be routine treatment, except in cases in which a separation of the symphysis is present. In such cases a high degree of lateral pressure is necessary, and is obtained by the use of the hammock without the spreader."

Noland further states this method of treatment gives high degree of ease and comfort to the patient, greatly simplifies nursing care, prevents a certain amount of muscular atrophy, allows for improved circulation throughout the pelvis, and allows easy access for care and dressings when the soft parts have been injured. He says:

"We believe that these fractures should be treated by traction and suspension for an average period of at least forty-two days. At the end of this time, the hammock is removed and the patient is allowed to move around about in bed for another week. The pelvis belt is applied immediately after the removal

Fig. 7. Case 16.—J. N. Fracture in two places of the right horizontal ramus of the pubis; fracture of the descending ramus of the pubis; fracture of the left pubic bone through the body; fracture of the sacrum at its lower right side near the sacro-iliac joint.

of the hammock. At the end of the seventh or eighth week, the patient is allowed out of bed and begins weight bearing with the aid of crutches.

As convalescence proceeds, daily hot tub baths and local heat are used. The patient should be kept under close observation for a long period. Judgment should be used in forcing these men back to work. In our early experience we were inclined to return them to work as early as possible, but frequently found that after a few days at work they were inclined to develop muscular spasm of the back and thighs, which necessitated return to bed with consequent lowering of morale. We attempt at all times to assure the patient that he has excellent prospects of recovery and that the long period of bed treatment is not because of the expectation of a permanent disability but to enable the fractures to be immobilized long enough for union to take place and healing of the soft structures."

From our own experience, the excellency of this method as outlined by Noland and Conwell appeals very strongly.

We quote from H. Winnett Orr of Nebraska, regarding his treatment of fractured pelvis, as follows:

"It is quite important in these injuries, as in most other fractures, to secure reposition of the fragments and to maintain this position during the entire period of healing. Wide abduction and traction (Whitman

position) are essential because they give just the kind of pull on the attached ligaments that will draw the injured parts of the pelvis into correct position. Fixed traction in a cast is the best method for maintaining length, and, finally, a double spica cast is required to immobilize the pelvis. Our technic in such cases is as follows:

"The traction is applied when the fracture is reduced and so immobilized or fixed in immobilizing apparatus that it and the fractured extremity remain undisturbed and unaltered until at least preliminary healing has taken place. The exact technic as employed by us is the following:

"Moleskin adhesive traction straps are applied to the limb, 3 inches wide and varying in length for fractures of the leg or thigh. The frayed ends of a strong, pliable rope are sewed into the distal ends of the straps to be used for traction purposes. The straps are applied to both sides of the limb well up to the site of the fracture and bandaged into place with muslin bandage.

"The patient is anesthetized on a fracture table; traction is instituted by a muslin bandage about the foot and the rope ends are attached to the traction device by which a pull is made and continued until the limbs are of equal length. The cast is applied in the usual manner, as far down on the extremity as the ankle. After the plaster is sufficiently set, traction having been continuously maintained by the foot bandage, the traction straps are cut off from the holding device, turned back against the leg portion of the cast, and embedded in the cast by several turns of plaster bandage, thus anchoring them and maintaining traction. When the plaster is set, the muslin bandage anchoring the foot is removed, and the foot wrapped with sheet wadding, and a cast applied with the foot at a right angle to the leg. Complete recovery and ability to be up and walk about may be expected by this method in 8 to 12 weeks."

In conclusion, let us reiterate that in pelvic fractures, as in skull fractures, the treatment of greatest importance is the treatment of the injury to the important soft structures and that we must diagnose these serious injuries to important soft structures early if we are to give life saving medical and surgical treatment at the right time, and that pelvic fractures with their accompanying serious injuries to important organs and high mortality rate will continue to increase unless the industries, the transportation companies and the people themselves in driving their own automobiles do everything possible to cut down the number of accidents.

CASE REPORTS

Case 1.—J. P., male, aged 21, single, was injured April 11, 1922. He was squeezed between a motor car and was rolled against a timber in an iron mine. The injury consisted of an excoriation of both hips just below the anterior inferior iliac spines. *X*-ray showed fracture across the right ilium from the anterior inferior iliac spine to the sacro-iliac synchondrosis without displacement. He was brought to the hospital in an ambulance. He remained in the hospital until May 19, 1922, thirty-eight days. He was confined in bed flat on his back for three weeks. Sandbags were used as supports. At the end of three weeks, he was allowed to get up on crutches and walk around the ward. At the time he left the hospital, he was able to walk outdoors on crutches. He was able to do light work on August 1, 1922, being disabled 111 days. He has worked in the mine constantly ever since at his regular job, function good.

Case 2.—C. N., male, aged 36, single, was injured May 25, 1922. He was brought to the hospital in an ambulance. He was injured in an iron mine by being squeezed between two cars. *X*-ray showed the following fractures: (1) fracture through the left ilium; (2) fracture through the descending ramus of the pubis. He was put to bed in the hospital flat on his back, the pelvis supported by sandbags. He complained at first of considerable pain in the pelvic region, pain on urination, and also some pain in the abdomen in the region of the stomach. There was no blood in the urine. He remained in bed in the hospital until July 3rd, when he was allowed to get up on crutches. After that he was up nearly every day on crutches and was discharged from the hospital on July 11th, having been in forty-seven days. He returned to work on October 6, 1922, disabled 134 days. At present time, he is working in the mine at his regular work. He feels well and function is excellent.

Case 3.—F. K., male, aged 37, married, was injured June 22, 1922, by falling down a skidway and being struck by rolling logs. He was brought to the hospital in fair condition. *X*-ray showed: (1) fracture of the horizontal ramus of the left pubis; (2) fracture of the descending ramus of the left pubis. He was put to bed in the hospital flat on his back with sandbag supports. He remained in the hospital forty-two days. He returned to work in one year, with a good functional result. He has been able to work since, but sometimes has a little pain and a slight limp.

Case 4.—J. V., male, aged 35, married, was injured August 18, 1922, in an iron mine by a cave-in. The patient was brought to the hospital vomiting and in a severe state of shock. He was at once treated for shock and catheterized, very little bloody urine being obtained. The abdomen was tense and hard; rupture of bladder was suspected. *X*-ray of the pelvis disclosed the following fractures: (1) fracture of the left ala of the ilium; (2) fracture of the horizontal ramus of the left pubis; (3) fracture of the horizontal ramus of the right pubis; (4) fracture of the descending ramus of the right pubis; (5) fracture of the transverse processes of the two lower lumbar vertebræ. He also had a hematoma in the right groin and a contusion of the right thigh. After two or three hours, when there was slight improvement in his con-

dition, he was prepared for operation and taken to the operating room. The abdomen was opened under local anesthesia. Blood and urine were found in the abdomen and an intraperitoneal tear in the fundus of the bladder, transverse and about one inch from side to side, was located. The mucosa was closed with catgut and the bladder wound closed by three rows of Lembert's silk sutures. A drainage tube was left in the abdomen down in the pelvis behind the bladder and a catheter was placed in the bladder through the urethra and secured in place. His condition was not materially worse after the operation. He failed to rally, however, after the shock of his injuries, gradually failed and died at 7:20 A. M., August 19, 1922, about thirteen hours after his injury. We did not feel that the operation in any way hastened his death.

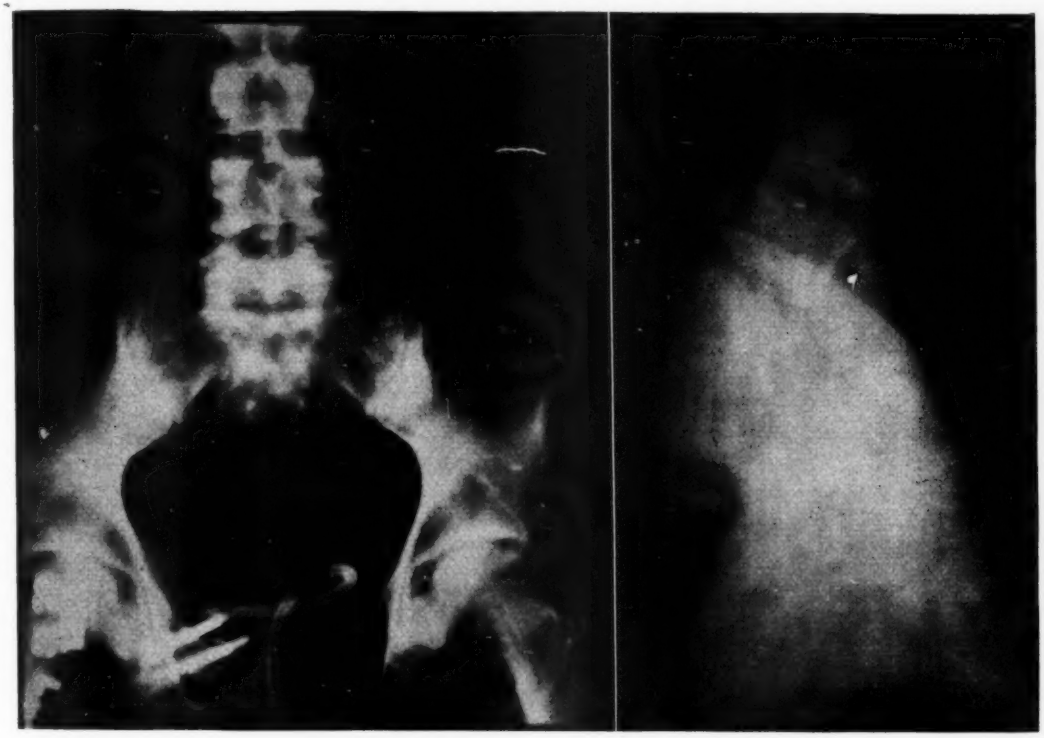

Fig. 8. Case 18.—M. R. Gunshot wound fracturing pelvis and spine with cord injury. Recovery.

Case 5.—J. O., male, aged 66, widower, was injured on January 25, 1923, while working in the woods, by a tree falling and striking him. He was brought to the hospital. His radial vessels were sclerotic, there was marked ecchymosis over the back and right hip posteriorly, crepitus marked over the crest of the right ilium, but no injury to the bladder, the urine being clear and normal. *X*-ray showed fracture through the crest and thin flat portion of the ala of the right ilium. He was given first aid and then sent to his doctor at his home in another town. No further history was recorded. Presumedly, he made a complete recovery.

Case 6.—E. M., female, aged 23, married, was injured on June 2, 1923, by a fall of thirty-five feet from a lookout tower. She and her husband, a forester, were in the tower when it was blown over by a windstorm. She was unable to walk but could crawl and helped her husband get out of the wreckage. He, though injured, sustaining a fractured shoulder, took a boat and went for help, leaving her alone. The accident happened at 1 P. M. They arrived at the hospital at 8 P. M. On arriving, she was put to bed and treated for some shock. It was necessary to catheterize her; the urine was examined and no blood was found. The patient was very tender over the lower abdomen. It was felt that she might have a perforation of the bladder or bowel. She did not vomit. *X*-ray picture taken showed a bilateral fracture of the pelvic bones, namely: (1) fracture of the horizontal ramus of the right pubic bone; (2) fracture of the descending ramus of the right pubic bone; (3) fracture of the horizontal ramus of the left pubic bone; (4) fracture of the descending ramus of the left pubic bone. Position was good and the patient was put to bed flat on her back and supported by sandbags. For several days following injury she had rather stormy symptoms, her lower abdomen being quite distended and

very tender but not markedly rigid. She had to be catheterized several times but no blood was found in the urine. Bowel movement was obtained by enemas. Symptoms gradually subsided but there was considerable ecchymosis in the perineal region; there was also a contusion of her left ankle, but *x*-ray showed no fracture. She was sitting up a little at the time before she left the hospital July 31, 1923, after a stay of fifty-nine days. She went home and went to bed. She was at home until August 17, 1923, when she had an attack of acute appendicitis. She was brought to the hospital and operated immediately, remained in the hospital eight days. She gradually recovered and began to walk and was able to attend to her household work on November 15, 1923. At present, she is doing her household work, feels well and has no pain or disability, function excellent.

Case 7.—A. L., male, aged 19, single, was brought to the hospital September 17, 1923. He had been injured in the region of the right kidney and right pelvis by being struck by a heavy steel bar while doing construction work. The patient was pale and cold, with a rapid, weak pulse and in a state of severe shock. All movements gave him great pain. He constantly moaned and seemed unable to answer questions. An *x*-ray taken at once of the lumbar spine and pelvis showed a fracture of the right crest of the ilium and fracture of the twelfth rib on the right side; no injury to the vertebra was shown. There was a considerable tumor-like swelling on the right side in the region of the kidney and lower back. The patient was catheterized and almost pure blood was obtained. It was thought the patient had an injured kidney, probably ruptured, with hemorrhage into the bladder and tissues surrounding the kidney, and a severe contusion of the back muscles. He was in poor condition for operation and immediate operation was not thought necessary. He was put to bed and treated for pain and shock. He had a rather stormy convalescence but gradually improved. No operation was performed. Blood disappeared from the urine entirely in about a month. His injury consisted of a fracture of the crest of the right ilium, fracture of the twelfth rib, probable injury to the right kidney (rupture) and ureter, severe contusion of the right side of the back, kidney region and lower back. He was up in a chair on October 24, 1923, walked about on crutches on November 2nd and left the hospital November 9th, being in the hospital for a period of fifty-eight days. Finally, he recovered with some deformity of the crest of the right ilium and a functionless kidney as reported by cystoscopic examination by a urologist. He is now working on the surface at the iron mines doing regular work, is well and healthy, does not complain of any pain or difficulty, walks well, has since married and has one child.

Case 8.—A. H., male, aged 47, single, was brought to the hospital April 29, 1924. He had been injured in an iron mine by being squeezed between a car and a post. He was in a bad state of shock. Examination showed severe contusion of the chest with traumatic asphyxia, his face being purple and eyes bloodshot. *X*-ray showed fracture of five or six ribs with considerable displacement and fracture of the left scapula with some displacement, fracture of the descending ramus of the pubis with no displacement. It looked as though he would live only a few hours. His pelvic injury was not nearly as serious as his chest injury. There was no blood in the urine. The first treatment was directed toward saving his life rather than attempting in any way to replace the broken bones. He gradually improved after a stormy convalescence. He left the hospital June 9, 1924, being able to walk on crutches, after forty-two days in the hospital. His back was very weak. He began light work March 9, 1925, being disabled 313 days. He went to his home in Finland, never quite fully recovered so that he could do his former work. The chest injury was the cause of this disability and not the pelvic fracture.

Case 9.—A. S., male, aged 54, married, was injured in an iron mine September 3, 1925. While stealing a ride in a tram car, he was struck by an overhanging chute. He was brought to the hospital in a state of shock, with considerable bleeding from an external wound over the right crest of the pelvis. *X*-ray showed the following: (1) compound fracture of the right crest of the ilium with displacement; (2) separation of the symphysis pubis; (3) separation of right sacro-iliac joint. Catheterization showed bloody urine. During the first week, he had to be catheterized at times and at times passed urine, usually with blood. The urine gradually cleared. Bleeding was probably due to the mucous membrane injury of the bladder and possibly other portions of the genito-urinary tract. He was treated by being placed flat on his back in bed and as soon as he was over his shock a wide canvas belt with buckles was applied, extending over the trochanters of the hip and brought together gradually by tightening up daily to bring the pubic bones in apposition and support the sacro-iliac joint. Fairly good apposition and position was obtained. He was in the hospital from September 3 to December 12, 1925, a total of 100 days. He commenced to move around some and walk on December 5th. At present he is alive and well and is working in the mine as a bell ringer. Before the injury he was a trammer. He started light work September 7, 1926. He complains of pain at times in the perineal region. He has had some permanent partial disability.

Case 10.—W. R., female, aged 39, married, was injured November 29, 1925, in an automobile accident. She was brought to the hospital semiconscious, cold and clammy, with a very weak pulse and in a state of shock. She had a large scalp wound on top of her head with considerable undermining of the tissues. The scalp wound was opened and explored; no skull fracture was found. There was concussion of the brain. She had a double fracture of the right clavicle, a small central piece about two inches long being entirely broken out and dislocated as shown by *x*-ray. She had numerous bruises over the body and an injury in the region of the right hip and pelvis. *X*-ray taken of lumbar spine, pelvis, and hip showed fracture of

the right ramus of the ischium, but no displacement. She passed blood in the urine for a week or ten days. The patient was put to bed in the hospital flat on her back and kept quiet. She recovered from the shock and went to her home on a stretcher December 8, 1925, having been in the hospital eight days. She was put to bed at home and remained in bed three months and then started to walk. At the end of four months, she began housework. She is now feeling well and has no trouble from the injury.

Fig. 9. Case 19.—J. S. Fracture of the horizontal ramus of the left pubis at its outer end and also at its inner end; fracture of the body of the left pubic bone; fracture of the descending ramus of the left pubic bone; fracture of the horizontal ramus of the right pubic bone at the outer end; fracture of the descending ramus, right pubis; some separation of the left sacro-iliac joint; fracture of the transverse processes of the lumbar spine. Death in three hours.

Case 11.—M. D., male, aged 28, married, was injured December 15, 1925, in an iron mine by being squeezed between a car and timber. The injury consisted of only a simple, irregular fracture of left ilium without displacement. He was put to bed in the hospital flat on his back with adhesive strapping of the pelvis. He left the hospital December 24, after nine days in the hospital. He went to his home to remain in bed. He was able, however, to walk around with crutches. January 25, 1926, he was walking with one crutch, but had some pain when walking. By February 8, 1925, he was doing well, walked without crutches but with a limp. April 5, 1926, he returned to work at the mine on light duty. At present he is well and healthy, doing a regular miner's work, with no pain or trouble from the fracture. He remained at home for a total of 110 days.

Case 12.—H. M., male, aged 46, single, was brought to the hospital, having suffered an injury in an automobile accident September 29, 1926. He complained of pain in the chest, difficulty in breathing, and his eyes were bloodshot and his face blue, somewhat like a traumatic asphyxia. He also had a pelvic injury. *X*-ray showed fracture of the descending ramus of the right pubis. He was put to bed in the hospital for a period of ten days, when he was allowed to go home and remain in bed. After he went home, he was in bed most of the time but sat up every day. Finally he began walking and went to work about four months after the injury. At present he is working in the mine and has no trouble nor disability.

Case 13.—R. G., male, aged 29, married, was injured April 4, 1927, by being squeezed and rolled between cars and timber in a mine. He was brought to the hospital in great pain and in some shock. *X*-ray showed fracture of the right ilium extending from the crest well down into the bone. General position was good. There was a large hematoma over the site of the injury. He was put to bed in the hospital, treated for mild shock, and sandbags were placed along the side of the pelvis and leg for support. The position of the fragments seemed good and no attempt was made to improve it. The patient did well in the hospital and was discharged April 26th, having been in the hospital twenty-two days. He was walking on crutches when he left. He was discharged for work July 6, 1927. At present he is working in the iron mines and has no trouble whatever.

Case 14.—M. B., male, aged 22, single, while out hunting November 27, 1927, was accidentally shot by the discharge of his companion's gun. He was brought to the hospital in a state of shock. Examination showed a young, healthy, robust man. He had lost considerable blood but his pulse was fair. He was in considerable pain, and a large gunshot wound was evident about 3 inches from the lower lumbar spine and 1 inch above the ilium in the left loin. *X*-ray showed a large number of shot buried and a fracture of the left ilium near the crest extending down to the sacro-iliac joint; also two transverse processes of the lower lum-

bar vertebræ fractured. The wound was cleaned up. Large numbers of shot, paper wads and other foreign material were removed. As there was considerable bleeding, the wound was packed with iodoform gauze. Patient was given 1,500 units of anti-tetanus serum and put to bed in the hospital. He had a rather stormy convalescence on account of considerable infection and some osteomyelitis with losses of pieces of bone. He left the hospital at the end of 70 days. When he left, he was able to walk. He came in for a long period of time for dressings. He started work May 1, 1928, having been disabled 154 days. He had an operation September 1, 1929. A large piece of bone was removed. Since then the wound has completely healed, and he is able to do any kind of work.

Case 15.—G. J., male, aged 47, married, was injured December 8, 1927, in an iron mine by a car of ore dumping over on him. He was brought to the hospital in a state of shock. He was a large muscular man and an old miner. Examination showed injury to the pelvis and a ruptured bladder was feared. He also had a traumatic asphyxia. His face was very much swollen and purplish in color. This extended down to the neck and upper part of the chest, especially to the front. He had some small hemorrhages under the skin and in the conjunctiva of the eyes. The pulse was fair. His greatest complaint was of pain in the pubic region. *X*-ray of the pelvis and lower dorsal and complete lumbar region showed nothing in the spine but a separation of the symphysis pubis and of the right sacro-iliac joint. The urine was a little smoky, had some red blood cells, a large amount of albumin, and gave a reaction for blood by the guaiac test. The bladder was filled with sterile boric acid solution and the full amount of fluid was returned. It was therefore felt that he did not have a ruptured bladder. The following day, the urine was clearing, was not so smoky and did not contain so much albumin. The urine gradually cleared up; no gross blood was seen at any time. The pubic bone was displaced upward on the right side. He was treated by extension traction and a wide belt placed around the trochanters with buckles tightened up gradually. He remained in the hospital until April 14, 1928, making a total of 128 days. At that time he was able to walk around with crutches. He began light work February 18, 1929, and has since continued to work.

Case 16.—J. N., male, aged 46, single, was injured by being struck by an automobile November 23, 1928. He was brought to the hospital. *X*-ray taken at once showed the following fractures of the pelvis: (1) fracture in two places of the right horizontal ramus of the pubis; (2) fracture of the descending ramus of the pubis; (3) fracture of the left pubic bone through the body; (4) fracture of the sacrum at its lower right side near the sacro-iliac joint. Position of the fragments was good except that there was a jamming down and impaction of the right horizontal ramus, which had been fractured in two places. This jamming down was at the pubic end. This patient was in good condition. There was no blood in the urine. There was considerable extravasation of blood in the tissues. Patient was placed flat in bed on his back and supported by sandbags. On January 5, 1929, he was able to walk and was allowed to go to the home of a friend, staying in the hospital 43 days. He returned to light work in about seven months or 210 days. At present he is working daily in an iron mine. He says that his pelvis does not feel quite secure and firm. There is considerable displacement of the fragment of the right horizontal ramus with some palpable deformity. This fragment possibly should have been operated on for better replacement.

Case 17.—S. W., male, aged 48, married, was injured March 9, 1929, by a cow jumping on him and knocking him down. He was brought to the hospital in good condition. *X*-ray showed fracture of left acetabulum with displacement upward of the head of the femur and about 0.5 inch shortening. He was put to bed in the hospital with traction, suspension and abduction. He later developed a foot drop, which, however, entirely cleared up. It was doubtless due to peroneal nerve injury. He remained in the hospital until May 4, 1929, a total of 56 days, when he was taken to his home and put to bed and extension reapplied. May 21st all extension was removed. The patient was in bed for a week, until May 29, 1929, when a walking caliper was applied. On July 9th the caliper was taken off; movements were excellent in all directions and there was about 0.5 inch shortening. At present time he is doing well, still limps some and walks with a cane.

Case 18.—M. R., male, aged 22, single, was accidentally shot July 30, 1929, and was brought to the hospital. It was found that a bullet had struck him on the right side about the region of the brim of the right ilium pretty well back, the bullet coming out on the opposite side. He lost considerable blood and was in poor condition. *X*-ray of the pelvis and lower spine region taken antero-posteriorly showed fracturing of the right ilium and lead scattered along the course of the bullet with injury to the spinous processes and laminæ of the fourth and fifth lumbar vertebræ. A lateral *x*-ray picture showed the same fractures. There was evidently some cord injury, as he was unable to empty the bladder and he had loss of power in his legs, although he was able to draw them up and move them quite well. The abdominal and cremasteric reflexes were present and sensation was present in both legs and feet. There was no reflex of the toes and he could move them only slightly. He remained in the hospital until August 10, 1929, when he was allowed to go to his sister's home. During the time he was in the hospital, he improved gradually. He was able in a few days to void and began to walk around on crutches after he went home. There was considerable infection of the wound and pieces of dead bone were removed at times. When last heard from he was walking with a cane and there was some discharge from the wound. He was seen June 14, 1930, when he could walk well using a cane but not without it. No knee

jerk could be obtained. The wound was completely healed. He thinks he will be able to work after a while.

Case 19.—J. S., male, aged 47, married, was injured April 8, 1930, by falling into a car and being struck by some ore. The patient was in a state of severe shock. *X*-ray showed: (1) fracture of the horizontal ramus of the left pubis at its outer end and also at its inner end; (2) fracture of the body of the left pubic bone; (3) fracture of the descending ramus of the left pubic bone; (4) fracture of the horizontal ramus of the right pubic bone at the outer end; (5) fracture of the descending ramus, right pubis; (6) some separation of the left sacro-iliac joint; (7) fracture of the transverse processes of the lumbar spine. The patient was in such a severe state of shock that he soon died, living only a little over three hours. No post-mortem was done and it was not known what internal injuries he may have had, such as ruptured bladder, bowel or blood vessels.

THE MANAGEMENT OF OPIUM ADDICTION

Many of the so-called specific cures have been given fair trial by critically minded observers, but the results have been consistently unconvincing. This applies to rationally conceived proposals as well as to exploited products like "Narcosan" or the alleged secret Kahle treatment discussed in the German medical press. The conclusion of a British reviewer seems to be justified that it is now realized more thoroughly than ever before that the major problem is not to free the addict from his drug but to keep him free. Morphine addiction is not characterized by physical deterioration or impairment of physical fitness. Herein lies the hope that rehabilitation by any process may be satisfactory so far as the physiologic functions are concerned. The mental and psychologic problems are not yet so easily disposed of. Relapse is common to all methods of treatment and the question as to whether the withdrawal of the alkaloid should be gradual rather than abrupt may be discussed in the light of many reports. British opinion, with a few notable exceptions, seems to be in favor of a reduction treatment as the routine method of cure. (Jour. A. M. A., September 13, 1930, p. 801.)

INTOXICATING EFFECT OF ACETYLSALICYLIC ACID AND COCA-COLA

A physician reports that young people in his community are using from 15 to 20 grains of acetylsalicylic acid (aspirin) dissolved in the soft drink, Coca-Cola, as an intoxicating beverage. The effects are due chiefly to central depression caused by the acetylsalicylic acid fortified possibly by a direct central stimulant action of caffeine and other constituents present in Coca-Cola. The actions of acetylsalicylic acid are similar to those of small doses of alcohol, or of a weak alcoholic beverage or of the barbitals or bromides. When acetylsalicylic acid is taken with a beverage like Coca-Cola, advertised for its exhilarating effects, which may be caused by a direct central stimulation by caffeine or other constituents of the latter, it is easy to see how the effects of one can be fortified by the other. The continued use of such a medicated beverage may result in undesirable effects of the same general nature as those of narcotic habituation. Here is a most insidious evil, the reinforcement of a popular and highly advertised beverage by a well advertised drug, both long suspected of dangerous tendencies. (Jour. A. M. A., November 1, 1930, p. 1366.)

SEROTHERAPY OF MEASLES AND POLIOMYELITIS

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THE annual death rate from measles is undoubtedly sufficiently high to warrant active efforts to accomplish its reduction. According to a report from London, measles during the past thirty years has become a more serious menace to life than scarlet fever, the former disease being responsible for a mortality rate of 251 per million as compared with 42 per million from the latter. In childhood, measles is responsible for about six per cent of deaths under three years of age, and for about 2.4 per cent of deaths during the first ten years of life. The mortality rate from measles may become exceedingly high following its appearance in communities which have been free from the disease for a number of years. This was illustrated by the epidemics which have occurred in certain Islands where the death rate has exceeded 25 per cent of the population. The serious potentialities of measles epidemic illustrate that such outbreaks cannot be viewed with complacency and emphasize the need of developing effective prophylactic and therapeutic measures.

For a number of years the contagium of measles has been known to be present in the blood, and also in the buccal and nasal secretions, particularly during the pre-eruptive and early stages of the disease. Attempts to isolate and identify the causative organism have not produced conclusive results, although these investigations have been conducted over a period of more than sixty years by many careful workers. During recent years the attention of investigators has been directed particularly toward the identification of either a filterable virus or a micrococcus as the possible cause of measles. Those who favor the micrococcus concept as the causative factor of measles (Ostrovsky ('97), Tunnicliff ('17), Caronia ('23), Cory and Day ('27) and others) however, apparently are individually considering somewhat dissimilar organisms falling in the group, and among themselves are unable to agree upon any one of these different strains of cocci as the sole and true etiology of measles. The demonstration that certain micrococci can be isolated from the blood of patients having this disease no doubt is of importance, but does not necessarily constitute proof that they are the etiologic agent.

Opposed to those who favor the micrococcus theory, there are a considerable number of investigators who consider a filterable virus to be the cause of measles. Those supporting this opinion include Hektoen ('05), Blake and Trask ('21), Degkwitz ('27) and others. To date, however, this filterable virus has not been isolated in pure culture.

At present one must feel that the etiology of measles is still disputed, and conclude further that the use of the various anti-measles sera for therapeutic, prophylactic and attenuating purposes other than convalescent or adult immune human serum probably should not be recommended for general use until more conclusive evidence is presented that the causative agent of measles has been definitely isolated, and that the various artificial sera are producing more constant results than have been reported in the literature.

THERAPEUTIC USE OF SERUM

The therapeutic administration of anti-measles serum is desirable in certain instances, particularly when the disease develops in a malignant form, and also when it arises as a complication of some previously existing acute and serious illness. Under such circumstances the administration of convalescent serum is of value in alleviating the severity of the disease and shortening its course. The first therapeutic use of convalescent serum was reported by Weisbecker in 1896. Since then Majoli ('15), Ribadeau-Dumas and Brissaud ('18), Terrien ('19), Maniel ('22), and others have employed the serum therapeutically with apparent benefit. Recently the production of animal anti-measles serum has been attempted, particularly by Tunnicliff and Hoyne ('26), but to date its therapeutic value is not sufficiently well established to justify recommending its general use. For the present, therefore,

the general therapeutic use of anti-measles serum should be limited to the administration of either convalescent serum or immune adult blood in rather large amounts as early in the course of the disease as possible.

PROPHYLACTIC USE OF ANTI-MEASLES SERUM OR PASSIVE IMMUNIZATION

The effectiveness of convalescent serum in the prevention of measles was first demonstrated by Cenci ('07), and since that time by numerous investigators, including Nicolle and Conseil ('18), Degkwitz ('20), Zinger ('24), Toomey ('26), Park and Freeman ('26), Bivings ('27), Boder ('29), Van Cleve ('30), Barenberg, Lewis and Messer ('30), and others. About 5 c.c. of serum collected between the tenth and ninetieth day of convalescence is sufficient to protect a young child. The passive immunity conferred in this manner, however, lasts only a few weeks. As pointed out in the bulletin of the New York Department of Health for January 25, 1930, "there are grave objections to any method which will entirely prevent measles passively, for this would lead to the gradual building up of a large population of non-immunes which would be decimated should the disease perchance be reintroduced." In communities that have been free from measles for years, and thus made up entirely of non-immunes, the disease is apt to develop a malignant form when newly introduced. For example, Panum ('27), reported that the appearance of measles in the Faroe Islands after the absence of the disease since 1781, resulted in 6,000 cases out of a population of 7,872. Also when measles was first introduced into the Sandwich Islands in 1775, within four months 40,000 of a population of 150,000 died. Likewise the introduction of measles in the Fiji Islands in 1874 resulted in the death of 20 per cent of the population.

Prophylactic use of convalescent measles serum, therefore, probably should be limited largely to prevent epidemics in institutions, or to prevent the development of the disease in weak and debilitated children in whom the occurrence of measles superimposed upon conditions previously existing might prove fatal. When the passive immunity is desired, the administration of 5 to 10 c.c. of serum collected in the first three months, and preferably in the first two or three weeks of convalescence is recommended. The serum should be given during the first four days after exposure, and repeated in twelve days (Barenberg, Lewis and Messer ('30)), if subsequent exposure seems probable. Aside from these special instances anti-measles serum probably should not be employed prophylactically, for the immunity conferred in this manner lasts only a short time and merely defers the date when the child will have the disease. When convalescent serum is not available, adult immune serum may be administered in larger amounts (60 c.c.).

THE ATTENUATION OF MEASLES BY IMMUNE SERUM

The greatest service physicians can render in the control of measles probably lies in the employment of measures which attenuate the disease rather than those that prevent it. In this manner a lasting immunity is developed by the patient from a mild attack of measles. This may be accomplished in a very practical and simple manner, through the administration of adult immune whole blood to the exposed children. According to the recommendation of the New York Board of Health, the blood should be obtained from the parent who has had measles and given intramuscularly, a procedure which does not require blood grouping. The dosage recommended for children under five years of age is 30 c.c., and double that amount for older patients. This permits the patient to develop a lasting immunity to the disease from a mild or attenuated complication-free attack of measles.

POLIOMYELITIS

Acute anterior poliomyelitis at first is an acute generalized systemic infection tending in the course of a few days to invade the central nervous system, following which the dreaded paralysis frequently occurs. In general there are three types of the disease, namely: the abortive, the non-paralytic and the paralytic type. In the abortive type it is assumed that the disease stops at some point in its development, failing for some unknown reason to proceed to localization in the central nervous system. Some feel that such abortive cases of poliomyelitis are very prevalent, and that consequently the majority of adult individuals have obtained an active immunity to the disease from mild attacks of this character. Since the diagnosis of the abortive type of the disease is based solely on symptoms, its preva-

lence remains largely a matter of conjecture. In cases where an invasion of the central nervous system occurs as demonstrated by physical signs and lumbar puncture, subsequent paralysis may or may not appear. This permits one, therefore, to recognize the clinically well established paralytic and non-paralytic forms of the disease, which unfortunately are indistinguishable at the outset. The physician, consequently, is compelled to administer serum for all cases of poliomyelitis recognized in the pre-paralytic stage. Since the serum must be given early in the disease if paralysis is to be prevented, the diagnosis of poliomyelitis in its pre-paralytic stage is of great importance.

At the onset the patient develops a fever, headache, and variable gasto-intestinal symptoms, including vomiting and diarrhea or constipation. The prostration seems to be greater than the temperature, which is usually under 102 F, would warrant. Although the general symptoms are fairly constant, they are not sufficiently characteristic to permit a recognition of the disease. A diagnosis of pre-paralytic poliomyelitis, therefore, must be made on the basis of the physical signs, which fortunately present a fairly uniform and significant picture in the early stage. One observes that the face is flushed except for the frequent presence of a definite circumoral pallor. In addition the throat is mildly reddened, and the pulse is usually accelerated out of proportion to the elevation in temperature. The remaining physical findings are limited practically to those which have to do with the nervous system. The patient may present varying degrees of hyperesthesia and slight clouding of the consciousness together with manifesting rather coarse tremors in certain instances. Of particular importance is the presence of a rigidity of the spine, which differs somewhat from the rigidity noted in meningitis. In infantile paralysis the head usually is not retracted, and although the neck is slightly rigid the rigidity seems to involve particularly the lower cervical, thoracic and lumbar rather than the upper cervical region. Thus in poliomyelitis the patient can usually flex the head on the neck at the atlanto-occipital joint, but is unable to bend the neck forward. As a result the head can be brought about half-way forward before resistance and pain are encountered. A general rigidity of the spine is a more constant and characteristic feature than stiffness of the neck. The spinal rigidity is so pronounced that in a sitting position the patient is unable to bend the head and trunk downward toward the knees. If the child bends forward at all, the flexion occurs almost entirely at the hips, while the spine remains straight and rigid. The deep reflexes are usually hyperactive in the pre-paralytic stage of the disease, and Kernig's sign as a rule is negative. The above signs require the performance of a spinal puncture in order to complete the diagnosis. The fluid obtained is usually under an increased pressure, and presents a very slight opalescence. The cell count usually ranges between 30 and 300, with the polymorphonuclear leukocytes predominating in the early stage of the disease. The polymorphonuclears, however, rapidly undergo a cytolysis, so that in a few days the cells are largely lymphocytes. In addition the globulin content of the spinal fluid is increased.

On the basis of the symptoms and physical findings as outlined above a diagnosis of pre-paralytic poliomyelitis probably is justified, particularly when care has been taken to exclude such conditions as meningismus, tuberculosis, lues, and lethargic encephalitis, conditions which might simulate infantile paralysis.

TREATMENT

The serum therapy of poliomyelitis has been limited largely to the administration of immune human serum. Rosenow ('30) has developed an anti-streptococcic serum for this purpose, and reports favorable results from its use. The outcome of pre-paralytic cases of poliomyelitis with respect to mortality and paralysis is so variable as to render it difficult to properly evaluate the efficacy of therapeutic measures instituted in such cases. This normal variability of the course of the disease in different individuals, therefore, has made many investigators skeptical of the alleged value of Rosenow's preparation. With regard to immune human serum there is apparently more unanimity of opinion as to its therapeutic effectiveness. Animal experiments have shown that convalescent serum neutralizes the virus of poliomyelitis and renders it innocuous, and also that the intrameningeal injection of the serum as late as twenty-four hours after the intracerebral injection of the virus prevents the development of paralytic disease. In addition beneficial results following the administration of immune human

serum in the early stages of the disease occurring in man has been reported by a considerable number of investigators. For the present, then, the use of convalescent human serum is recommended for therapeutic purposes. The serum preferably should be administered as early in the disease as possible, giving 20 to 30 c.c. intraspinally and 50 c.c. intravenously or intramuscularly. If necessary the entire dosage may be given intravenously, although it is probably more advisable for a certain amount of the serum to be given intraspinally. Favorable results have been reported by Shaw and Thelander ('28) even from the intramuscular administration of the serum. Although the best results are obtained where the serum is given in the pre-paralytic stage of the disease, one should not hesitate to administer it after paralysis occurs, attempting thereby either to prevent a further extension of the paralysis or to promote as complete a recovery as is possible to obtain.

In the presence of severe outbreaks of epidemic poliomyelitis, convalescent human serum may be used to afford passive protection to children exposed to the disease. Although the optimum amount of serum to be used for prophylactic purposes has not been definitely established, Flexner and Stewart[28] suggest a subcutaneous dosage of 10 c.c. for young children and 20 c.c. for older children and adults. A repetition of the injection at the end of four to six weeks is suggested, should the epidemic outbreak become severe and endure for several weeks.

The main objection to the use of convalescent human serum for therapeutic and prophylactic purposes is the difficulty of obtaining it. Partly for this reason it probably will be used more generally at times when there are a considerable number of cases of the disease appearing in a community, rather than when the outbreaks are limited to sporadic instances.

At present there is little to be said concerning active immunization in poliomyelitis, aside from calling attention to the fact that monkeys have been rendered immune to the disease through intradermal injection of the virus.

SUMMARY

1. Convalescent serum or immune adult blood when used in rather large amounts may be administered beneficially for therapeutic purposes in instances where measles develops in a malignant form, and also when it arises superimposed on some previously existing acute and serious illness.

2. For prophylactic purposes the use of convalescent measles serum probably should be limited largely to prevent epidemics in institutions, and also to prevent the development of the disease in weak and debilitated children. Five to ten c.c. of convalescent serum should be given within the first four days after exposure. When convalescent serum is not available, adult immune serum may be used, but should be given in rather large amounts.

3. The greatest service physicians can render in the control of measles involves an attenuation of the disease, thereby permitting the patient to develop a permanent immunity to measles from a mild complication-free attack. This may be accomplished by using immune adult blood, giving 30 c.c. intramuscularly to children under five years, and double that amount to older patients.

4. When dealing with poliomyelitis the therapeutic administration of convalescent human serum, preferably in the pre-paralytic stage of the disease, is recommended. For this purpose give 20 to 30 c.c. intraspinally together with 50 to 75 c.c. intravenously. Furthermore, one should not hesitate to administer the serum after paralysis occurs, attempting, thereby, either to prevent a further extension of the paralysis or to promote as complete a recovery as possible.

5. In the presence of severe epidemics convalescent serum may be used to afford passive protection to exposed children, giving 10 c.c. subcutaneously to young children, and double that amount to older patients. A repetition of the injection at the end of four to six weeks is suggested should the epidemic outbreak continue in severity for several weeks.

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SODIUM 2-OXO-5-IODO-PYRIDINE-N-ACETATE (INTRODUCED AS UROSELECTAN)

The Council on Pharmacy and Chemistry publishes a preliminary report on sodium 2-oxo-5-iodo-pyridine-N-acetate (also designated as sodium-5-iodo-2-pyridon-N-acetate), a new substance for use in the radiographic visualization of the urinary tract which was introduced under the name Uroselectan. The Council reports that the product is a definite chemical substance which promises to be an advance in the radiographic visualization of the urinary tract, though its indications, advantages and limitations are at the present time not fully established. The Council considers the product suitable for experimental use by those who are versed in the technic of urologic examinations. It publishes a concise and carefully considered statement of the introduction of the drug and the evidence for its use, prepared for the Council by Drs. Braasch and Bumpus. (Jour. A. M. A., November 8, 1930, p. 1425.)

THE ETIOLOGY OF GINGER PARALYSIS

Within the past year a peculiar form of paralysis has afflicted many persons, particularly throughout the Midwestern or Southwestern states. Evidence has accumulated that the malady is closely associated with the drinking of fluidextract of ginger. The latter has been sold extensively since the introduction of the Volstead Act, for beverage purposes, because of a ruling of the Prohibition Bureau to the effect that the official fluidextract of ginger is a non-potable beverage, thus removing the restriction from its sale. It was concluded from the evidence that an adulterated and poisoned product was being circulated last winter. An investigation was undertaken by the United States Public Health Service and this has shown that the paralysis was caused by tricresyl phosphate present in some fluidextracts of ginger as an adulterant. (Jour. A. M. A., November 29, 1930, p. 1672.)

IMMUNOLOGY OF DIPHTHERIA AND SCARLET FEVER*

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WITH the steady advance in the specific knowledge of disease, readaptations and readjustments of thought and action must keep pace. It is our purpose here to present an appraisal of present day opinion and practice in the specific control of diphtheria and scarlet fever.

Little need be said about the use of specific serum or antitoxin for the cure of diphtheria. It is sufficient to say that it must be used early and in adequate dosage to neutralize all of the toxin of the infection if its maximum value is to be realized. Schick recommends from 100 units per kilogram in the mild cases to 500 units in the most severe cases. It should be given all in one dose and not in small repeated doses except in cases to be mentioned later where desensitization must be carried out. Naturally rapidity of action depends upon the promptness with which the antitoxin gets into the blood stream. Therefore in the most critical cases it should be introduced intravenously. In those slightly less serious or in whom intravenous injection is impossible, intraperitoneal injection affords a more rapid means of introduction than the third way, namely, the intramuscular route. The serum should never be introduced subcutaneously, except for purposes of desensitization, because of the slow absorption by this route.

It has been common practice to use antitoxin for prophylactic so-called passive immunization in cases of actual or suspected exposure. While occasionally justifiable, modern medical practice demands that we consider the possible costs of such action. The immunity so induced lasts no more than two or three weeks, frequently subjects the patient to a discomforting serum reaction and leaves him sensitized to horse serum, thus increasing the hazards in case a future necessity might arise for the use of an antitoxin. In the great majority of cases it would be better practice to postpone the use of immune serum until careful supervision shows suspicious symptoms of the disease or until positive nose or throat cultures have been obtained. Final decision of policy in this regard must, of course, rest with the individual circumstances of each case.

Present advance in the control of diphtheria is being made mainly along the lines of active immunization by means of vaccination. The success of this procedure is made possible by the development of an easily applied test of susceptibility to the disease. This test, named the Schick test after the man who developed it, consists of the intracutaneous injection of a measured amount of diphtheria toxin (1/50 M.L.D.) contained in a suitable medium. While extreme emphasis is placed upon an exact technic in its performance by most writers, as an actual matter of fact the test has a sufficiently wide margin of safety, as demonstrated by Dudley, to be extremely valuable even in the hands of a novice. The points usually emphasized are these: that exactly the correct amount of toxin must be introduced, that it must be injected intradermally and not too deeply, and that the toxin must be of proper potency. While all of these points are important, Dudley has shown that a variation of 25% either way in the amount of toxin injected would not weaken the value of the test too greatly and that even subcutaneous injection will usually afford accurate information, though perhaps a little more difficult of interpretation. The one important requirement is that the toxin shall be of proper potency. In this respect the clinician is largely at the mercy of the manufacturer and must accept his claims, but at least the physician can use meticulous care in carrying out instructions given in the package. This is especially important as regards the promptness with which the toxin is used after dilution. It must be used within twenty-four hours and preferably within a very few hours after dilution.

Unless an individual has substances in his blood to counteract the toxin injected he will develop a characteristic inflammatory reaction at the site of the injection. This reaction is termed a positive Schick, and it will be noted unless the subject has as much as 1/30 unit of antitoxin per c.c. of serum. Approximately this amount of antitoxin in the blood is required to neutralize the amount of toxin injected and has been found by clinical experience to be sufficient to protect

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the individual against any ordinary contact with the disease. Hence a negative Schick reactor has this much or more antitoxin in the blood and is essentially immune.

The interpretation of the Schick test offers little difficulty in the majority of cases. Dudley describes a typical positive reaction as follows: " . . . in about 24 hours, or earlier, . . . a red, almost scarlet, patch about an inch in diameter appears, which is generally elliptical rather than circular, the major axis of the ellipse being in line with the limb. In four or five days the reaction, after first becoming more intense, begins to fade and desquamate, and become covered with bran-like flakes of cracked superficial epithelium, which gives to the reaction a most characteristic appearance at the end of a week. The reaction disappears slowly, and remains evident for a month or more as a pigmented patch of variable tint. A certain amount of induration accompanies the color reaction during the active stage. Variations either way from this usual reaction may occur, on the side of more intensity up to the point where vesiculation and superficial ulceration may be present, and on the side of lesser intensity to the point where nothing in the way of pigmentation or desquamation may be observed. The former indicates increased susceptibility and the latter a partial immunity to diphtheria."

Some confusion in the reading of the tests may be met with on account of a pseudo-Schick reaction. While this reaction has certain qualities which usually identify it to the experienced, the casual observer must rely upon the duration of the inflammation for its recognition. According to Dickenson only 4.5 per cent of pseudo-Schick reactions are visible at the end of a week. Park says that pseudo-reactions constitute about 15 per cent of the reactions in this country. Thus in not more than two-thirds of one per cent of the reacting cases would the pseudo-Schick be even visible at the end of a week. If we read our tests after seven days then, in only a very small number of cases should there be any confusion and these will usually be cases in which both a pseudo and positive Schick have occurred, the so-called combined reaction.

The discovery of the Schick test has opened the way for extensive minute study of diphtheria. As a result it has been found that almost all infants are immune to diphtheria at birth and that rather rapidly during the latter part of the first year this immunity is lost. Breast feeding retards this loss of immunity. After one year of age the incidence of immunity as shown by the number of negative Schick reactions begins gradually to increase. The congenital immunity is thought to have been passively transferred to the infant before birth through the placenta and for some time later through the breast milk. The later rise is thought to be actively acquired as a result of repeated subclinical infections. Hence the number of persons exhibiting this natural immunity increases with age and directly in proportion to the existence of those factors which favor exposure such as crowded living, etc. Thus it is that those persons living in rural communities are less apt to have acquired an immunity by the time they have reached adult life.

The natural sequel of such observation was the attempted artificial production of an active immunity by means of vaccination. This has proven so successful that it is now one of the most important public health measures. It has become a moral responsibility to the medical profession so well recognized that if physicians do not accept it this important health measure will, rightly, be taken out of their hands altogether.

The preparation most widely used in this country for this purpose is a solution of diphtheria toxin partially neutralized by the addition of antitoxin. It is necessary to neutralize a part of the toxin in order that a sufficient amount may be injected to bring about an immunity reaction. Naked toxin is too toxic to be used unbuffered.

Three inoculations of 1 c.c. each are given at weekly intervals. Following these injections a gradually developing immunity appears, due to the presence of antitoxin in the blood, which by the time six months have passed will have increased sufficiently to render the Schick test negative in about 75 or 80 per cent of cases.

Positive reactors should at this time receive a second series of injections, following which most will have become Schick-negative. Persons still remaining Schick-positive should be given a third series of inoculations, in spite of which a small number may still remain Schick-positive, apparently unable to produce antitoxin. There is reason to believe that the duration of the immunity so induced is permanent. Park has shown that 90 per cent have shown a permanent immunity

over a period of nine years. It seems probable that the incidence of permanent immunity might in some measure be influenced by the occurrence of minor exposures to the disease. In those cases which again become Schick positive it is probable that they retain some degree of immunity in that they seem to retain an ability to form antitoxin rapidly when necessary. In these cases a very small stimulus will cause a rapid disappearance of the Schick test.

The greatest drawback to the use of this product for immunizing purposes is the fact that by using it one is sensitizing the individual to horse serum and thus increasing the hazards of any future emergency necessitating the use of horse serum. Park and several other men of wide experience feel that this factor is negligible but numerous reports indicate that this is indeed a factor that must be considered. The work of Gordon and Creswell is especially convincing, and it is hardly believable that any careful practitioner would willingly advise running this risk in such a wholesale manner when the uses of horse serum in present day medicine are becoming so rapidly increased. This must be thought of as a definite objection to the use of toxin-antitoxin for immunization to diphtheria, not great enough to preclude its use so long as no equally good substitute was available but enough so to stimulate search for such a substitute.

This was a factor in the prolonged delay in the usage of toxin-antitoxin in France. It probably was largely responsible for the development by Ramon of a vaccine which he called antitoxin that was rendered non-toxic by treatment with formaldehyde and heat. This treatment destroys the poisonous quality of the diphtheria toxin but leaves its immunizing properties intact. Ramon and many others have used it extensively in France and find it not only safe for injection, but also much superior to toxin-antitoxin in immunizing value. Since it contains no horse serum it has no sensitizing qualities. Quite a number of American investigators have also used it and substantiate all of the claims of Ramon and his co-workers. Ramon's method called for the subcutaneous injection of three doses; the first 0.5 c.c., the second 1 c.c. given three weeks later and the third 1 or 1.5 c.c. after another two weeks. Dick and Dick found a two week interval between all three injections very satisfactory. Rarely under the age of eight but with increasing frequency as age advances from this point, reactions of varying intensity occur with the use of toxoid. These reactions are principally local and usually unimportant from a general standpoint. They seem to depend upon a sensitization to some of the protein substances of bacterial or other origin contained within the injected fluid. They are not reactions to the diphtheria toxin. Thus they result from individual sensitivity to the same substances that are responsible for the pseudo-Schick reaction, and the Dicks maintain that if such a reaction occurs in the preliminary Schick test it is best to begin immunization with .1 to .25 c.c. of the toxoid. Results with toxoid yield from 95 to 100 per cent immunity after three injections as compared with from 75 to 80 per cent with toxin-antitoxin. Furthermore the time necessary for the production of immunity is much less, the Schick becoming negative usually in from five to six weeks.

In an effort to eliminate the sensitizing properties of toxin-antitoxin, Larson found that he could render diphtheria toxin innocuous without destroying its immunizing quality by mixing it with a solution of castor oil soap (sodium ricinoleate). He has had marked success in immunizing against diphtheria with this product. At the Gillette State Hospital for Crippled Children, where it has been used combined in the same solution with scarlet toxin for the past four years with an average daily population of 230 children, there have been six clinical cases of diphtheria with an additional 39 positive culture cases without clinical symptoms.

Twenty-five out of twenty-seven of these 39 that were so tested had virulent organisms in the cultures. Compared with the three year period preceding, when 252 positive culture cases occurred, most of them representing clinical diphtheria, this is most strikingly successful control.

It becomes evident when one considers the question that the development of the Schick test was the basic principle upon which all the recent advance in the understanding and control of diphtheria was based. The knowledge so attained was theoretically transferable to any contagious disease whose causative agent was known and in which the symptoms were produced through the agency of a soluble toxin. Thus it was that when the Dicks had demonstrated the organism of scarlet fever to be a form of hemolytic streptococcus liberating a soluble toxin

which was responsible for the immediate symptoms of the disease, the path to rapid progress was clearly defined. The skin test for susceptibility, congenital immunity followed by increasing susceptibility with subsequent gradually acquired natural immunity as age advances, vaccination for the production of artificial immunity and finally antitoxin for the control of the disease were all suggested at the start.

In the use of the skin test considerable difficulty has been experienced by many observers. The Dicks defend the test very vigorously and say that while it is very exacting it affords results that are very reliable when all details as to procedure are carefully carried out. They claim 100 per cent reliability for a total of more than 35,000 persons. The method is similar to that used in the Schick test. One-tenth c.c. of a solution of toxin of such a titre that this amount represents the rather arbitrary unit of one skin test dose is injected into the skin of the forearm. In controlling artificial immunity the Dicks make another injection of twice this amount on the other arm. The test must be read under a strong light after from 18 to 24 hours. Any redness as large as 10 mm. in any diameter is regarded as positive. No induration is required and tests read after 24 hours are not reliable.

Using this test the Dicks have traced a curve of natural immunity essentially similar to that already described for diphtheria. As in this disease, the percentage of immunity in a given group is proportional to the opportunity for repeated subclinical exposure.

As to the artificial production of active immunity by vaccination the problem is somewhat different than in diphtheria. The streptococcus of scarlet fever produces its deleterious effects not only through the agency of a soluble toxin but also through actual invasion of the tissues, and many of the worst elements of the disease are a result of the latter quality. Present methods of study are not effective in measuring the antibacterial elements of immunity but are confined to the antitoxic factors. With even this handicap, however, the Dicks claim a tremendous success in artificial immunity, again 100 per cent efficiency. They inject subcutaneously graduated amounts of naked toxin at weekly intervals, increasing from 500 skin test doses in the first injection up to from 80,000 to 100,000 skin test doses in the fifth or last inoculation. If the Dick test two weeks later is still positive the fifth dose is repeated.

Others have found the reactions caused by the Dick technic too severe for general usage. The originators themselves admit that about 10 per cent of the injections are accompanied by general reactions, though how severe these are is omitted from the summary I am quoting. They do, however, state that no permanent harm has resulted from active immunization of 12,775 susceptible individuals.

The effort is constantly being made to find a method of immunization that is more practical than one requiring five injections and less severe in its immediate effects. Peters and Allison have used a method by which they spray the toxin into the nose, and have obtained rather a remarkable degree of success. No reaction whatever occurs during the immunizing process. Several workers have used scarlet toxoid successfully. Here also disagreeable reactions are lacking. The application of the toxin in ointment form to the skin represents another effort at simplifying the process of immunization.

In 1926 Larson began immunizing against scarlet fever with a toxin buffered, or rendered relatively non-toxic, by the addition of sodium ricinoleate. Certain advantages were noted in a toxin so protected over the naked toxin as used by the Dicks. Larger doses could be employed without bringing about disagreeable reactions and the degree of immunity produced seemed to be definitely greater than that produced with a like amount of toxin unbuffered. Later this toxin was mixed with a suspension of the organisms of scarlet fever, thus using not only a toxin but also the organism for immunity production. The Dicks have viciously attacked this method of immunization as totally incompetent and misrepresented, giving a wholly false sense of security because of its total failure to accomplish its ends.

Since November 1, 1926, the Board of Health of the City of Minneapolis has been using this vaccine, combined with diphtheria toxin in the same dose. From that date until August 31, 1929, a total of 24,212 injections had been made. A complete course of treatment, three doses, had been given to 6,953 children and incomplete courses, one or two doses, to 2,205 more, making a total of 9,158 children immunized or partially so. It was estimated that 4,000 more were

treated by this method by private physicians. The total number of children in Minneapolis between the ages of one and sixteen was estimated at 105,000. During the observation period 3,973 cases of scarlet fever were reported. Of this number only 55 were in children who had received the ricinoleated vaccine. Scarlet fever was thus ten times as common among those children who had not received Larson's vaccine as among those who had. Parks claims for toxin-antitoxin only a reduction to one-fifth the incidence in those who have not received such vaccination against diphtheria. From this experiment it would seem that practically the ricinoleated scarlet fever toxin is very efficient in controlling scarlet fever.

Again, at the Gillette State Hospital for Crippled Children, where this vaccine has also been used in combination with the diphtheria toxin, from May, 1926, to July, 1930, only eleven cases of scarlet fever occurred. Of these eleven, five occurred within a week after admission to the hospital. Thus only six cases of scarlet fever developed in patients that had been in the hospital long enough to be immunized. During this time twelve adult attendants who had not been immunized developed the disease. Any of the attendants or of the eleven patients would have been capable of starting an epidemic if the number of susceptibles had not been very low. During this period 75 per cent of the population came from rural communities.

The duration of artificially produced immunity is a question that can not as yet be settled. The Dicks claim that retests made at the end of one, two and three years indicate that 90 per cent of patients who are immunized to the point of entirely negative skin reactions, retain their immunity. Between 5 and 9 per cent require second immunization. It seems fair speculation that the duration of such immunity might in some measure at least be influenced by the occurrence of slight subclinical infections and that thus the conditions of living, such as crowding, etc., might play the same role here as they do in the development of natural immunity.

The same factors that complicate the problem of artificial immunization render the problem of antitoxin more difficult. For maximum efficiency a serum that is both antitoxic and antibacterial should be produced. This has led to two principal methods in the production of scarlet fever serum. The first is made by injecting the horse with toxin alone, thus stimulating the production of a pure antitoxin. In the second method an attempt is made to produce an antibacterial serum as well by inoculating live organisms into an agar plant that has been placed within the body of the animal. Thus not only is the horse forced to produce an antitoxin but also reactionary bodies against the bacteria themselves. No marked superiority has as yet been demonstrated for either method, though at present most commercial serums are produced in the latter manner.

There is little doubt but that scarlet fever antitoxin is effective in controlling the immediate symptoms of the disease. Here, as in diphtheria, it should be given early for maximum effect. There is much more generally expressed doubt as to the value of the serum in preventing complications. The latter, it will be noted, are usually a result of bacterial invasion and not of purely toxic origin. On the other hand certain enthusiastic supporters of the serum treatment insist that the complications are very materially reduced by the use of serum. Still other writers hold that the serum does not affect the incidence of complications in mildly toxic cases, but that it does in the severe types. Thus two general opinions are to be found among writers on the subject: one that the serum should be used as soon as scarlet fever can be diagnosed, regardless of severity, or even as soon as scarlet fever is suspected (Dick); and the other that would limit its use to those cases that show marked toxemia at the start. The latter opinion is certainly the more conservative and it seems to me that conservatism has virtue in this time of wholesale serum injection. Mild scarlet fever is not a severe disease. Even moderate grades of serum disease are much more discomforting, and serum reactions are particularly common following the administration of scarlet fever serum. It is true that serious complications may supervene even following the mildest cases of scarlet fever, but until it is thoroughly proven that the serum is of considerable benefit in preventing such accidents in the mild cases, we are not justified in subjecting the patient to the hazards and discomforts that so frequently follow its use. There can, however, be no doubt that it should be used in the moderately severe and severe cases. The use of scarlet fever serum for prophylaxis in case of exposure is mentioned only to be con-

demned. It should never be used then. Before the discovery of antitoxin, convalescent serum was used in the treatment of scarlet fever with some degree of success. The obvious difficulties encountered in procuring it when needed greatly limited its use in general practice.

When scarlet fever antitoxin first appeared, it was thought that it would be useful as a means of demonstrating the skin blanching phenomenon (Schultz-Charlton test) and thus aid in clearing the diagnosis in certain doubtful skin eruptions. Toomey and his co-workers have shown that most of the serums are useless for this purpose, some even giving rise to an increased redness at the site of injection. These authors aptly suggest that if the blanching test is dependent upon the antitoxin content of convalescent serum, then most of the commercial serums are devoid of antitoxin.

It is usually a safe maxim that nothing is acquired without a price being paid for it. This is true of the progress that is being made against the two diseases under discussion. New problems arise, by-products of the new methods employed, which while less of a menace than the original difficulty must nevertheless be dealt with. Two problems which pertain especially here are that of the carrier and that of sensitization to horse serum. The first of these has been recognized as a formidable obstacle in the control of diphtheria for many years. It bids fair to become a much greater problem as the number of immunes increases. This is especially true in institutions where it is possible for a large number of inmates to become carriers after immunization in such an insidious manner as to produce no symptoms whatsoever. Only the closest imaginable control, combined with frequent cultures, will cope at all with this menace. While the carrier has long been recognized as a menace as regards scarlet fever, the lack of method for identification has limited the problem to one of surmise. The precise methods—culture, etc.—which are bound to develop from the increasing knowledge of this disease, give promise of duplicating for scarlet fever what is happening in the case of diphtheria. Much must yet be done in the control of these diseases along the line of prevention and cure of the carrier.

Even more important is the problem of sensitization to horse serum. While some of those who deal with this factor in the immunization of thousands of cases are inclined to think lightly of it, the physician who encounters the single case of sensitization is apt to regard this phase of the subject more seriously. He realizes that a single death or near death from the giving of a serum, were it traced to methods employed in the efforts to prevent disease, would do more damage to the cause of prophylaxis than a dozen deaths from the lack of it could rectify. Such is the psychology of the uninformed. With the present trend in medical practice it will not be long until a great majority of individuals will not only have become sensitized to horse serum, but will have received one or more shock doses before they have run the gamut of common diseases with their prophylactic and curative procedures. Certain observations seem to indicate that the severity of the reactions increases with the number of injections. While this phase of the subject should not be overemphasized, at the same time it is not wise to totally disregard it. A sensitized state may hinder or delay the giving of therapeutic serum in circumstances where this delay is costly, and until we understand more about the subject of protein sensitization, or anaphylaxis, and are better able to cope with it, we will be wise if we avoid any unnecessary contact with it.

Regardless of what policy we adopt, we probably shall always have with us the sensitized individual to whom it is absolutely necessary to give horse serum in some form. Such individuals may be naturally sensitive (the more dangerous type) or they may have been sensitized through a previous injection of horse serum. Therefore, before serum is given, careful inquiry should always be made as to whether or not any form of serum had ever before been given. If not, it is wise to inquire as to the occurrence of asthma, especially horse asthma, hay fever, eczema, urticaria or other manifestations of allergy in either the patient or his near relatives. Positive answers to either question obligate the physician to determine whether the patient is sensitized. Even though no such history is obtained, it would still be the more conservative procedure to test the patient for sensitization before introducing a large amount of serum.

This is especially true when the injection has to be made either intravenously, intraperitoneally or intraspinally.

Mackenzie and Hanger have covered this sub-

ject in magnificent detail and their ideas on technic are as follows: To determine sensitivity, a minute amount, not more than 0.05 c.c., of a 1 to 10 dilution of serum is injected into the skin of the forearm. They advise the injection of a like amount of human serum or normal saline at the same time as a control. If the wheal induced by the injection enlarges and becomes surrounded by a zone of erythema in from ten to twenty minutes the patient is sensitive, provided the control does not behave the same way. In extremely sensitive individuals an enormous reaction may be induced. Pseudopod-like projections at the edges of the wheal occur with the higher degrees of sensitization. They advise the dilution of the serum because in this way false reactions do not occur. One can not depend altogether upon the character of the skin reaction as an indication of the degree of sensitivity of the individual. Severe reactions are not unknown, they say, in the absence of a positive cutaneous test. Therefore, even though the test may be negative, it is wise to proceed cautiously whenever a suspicious history of sensitization is obtained.

In the sensitized person desensitization is necessary. These authors recommend the initial injection of .01 c.c. of serum suspended in saline and given subcutaneously. Every half hour this injection may be repeated, doubling the dose of serum each time unless toxic symptoms occur. In such case, the next dose should be of the same amount as the preceding in order not to proceed too rapidly. When as much as 1.0 c.c. of serum has been tolerated at one time subcutaneously the next dose may be given intravenously if this method of injection be necessary. This should be reduced to .1 c.c. in saline and may be doubled each twenty minutes thereafter until the full therapeutic dose has been injected.

In cases where intraspinous injection is to be given, one should continue with the intravenous injections until 10 c.c. are tolerated. Then, after the proper interval, a like amount may be cautiously injected into the spinal canal.

Occasionally individuals are encountered in whom it seems to be impossible to bring about sufficient desensitization. Still others may be desensitized more rapidly than this technic prescribes if no reactions occur after the early doses. Almost all individuals, however, can be desensitized to the point where serum in therapeutic dosage can be safely given.

CEREBROSPINAL MENINGITIS*

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CEREBROSPINAL, epidemic, or meningococcic meningitis, a disease constantly present in all large communities and often epidemic in character, continues to present difficulties in control despite our knowledge concerning it. The purpose of this paper is to summarize recent work on this subject.

Cerebrospinal meningitis is essentially a disease of children, though it may occur at any age. In Neal's series of cases in New York City, 70 per cent occurred during the first ten years of life. A great many cases appear during the first year of life and at this age it is the most common type of meningitis.

Nearly all epidemics occur during the winter and spring months; overcrowding is a factor in spreading the disease. Apparently it is a disease to which most persons have a natural immunity; multiple cases in a family are uncommon. Leukowicz states that the meningococcus is really a very slightly pathogenic organism, but its feeble virulence is largely counterbalanced by its remarkable power of multiplying, which enables it to appear at times in blood films or to provoke extensive skin rashes in septicemic cases. As soon as the body develops some general immunity, the meningococci in the circulation are impaired in vitality and soon are completely destroyed. However, a marked difference in virulence in various epidemics occurs.

DISSEMINATION

Meningitis is disseminated through the medium of healthy carriers. The meningococcus is found at some stage in the nose and throat of those ill with the disease in over 90 per cent of cases. It usually disappears in from five to fourteen days. Mathers and Herrold found that among 140 immediate contacts (tent-mates) of cases of meningitis, 27 per cent were carriers. Seven of these were persistent carriers. Of 15,257 men, members of organizations in which meningitis had occurred, 44 per cent yielded positive meningococcic cultures. They found two types of carriers: temporary and persistent. The persistent carriers, individuals yielding positive naso-pharyngeal cultures over a long period of time, were few in number and only rarely developed meningitis. In the cultures from these carriers the organisms were present in predominating numbers. The temporary carriers were more numerous, but their cultures showed few meningococci, which persisted only over a short period. The biologic type of meningococcus predominant in the camp epidemic at the time was identical with that among the chronic carriers; also the type found in the cases and in the immediate contacts was the same. These facts suggest that there is a close relation between cases of epidemic meningitis and carriers. They feel that the persistent or chronic carrier is especially dangerous. Gates reports that specific agglutinins have been found in the blood serum of chronic carriers, evidence that their relative immunity may be due to these antibodies.

MORTALITY RATE

The general mortality rate from this disease has been reduced from approximately 70 per cent before the introduction of serum to variously estimated figures of 23 to 35 per cent, since. These figures include cases in infants under one year of age. This high death rate may be due to several factors: (1) An extremely virulent organism causing a fulminating meningitis; (2) an organism of different type than those used in producing the serum; (3) late recognition of the disease. The last mentioned is certainly the most common. Meningitis occurring in infancy, however, is still carrying a decidedly higher mortality rate, approximately 45 to 50 per cent. The causes for this higher mortality are late recognition of the disease, and lack of resistance to the organism.

Gates has injected volunteers with a meningococcic vaccine and later demonstrated specific meningococcic agglutinins in their blood serum. I have come across no other reference regarding active immunization against the disease.

SYMPTOMATOLOGY

As to the disease itself there are four types:

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(1) The common type, usually seen in older children or adults; (2) the fulminating type; (3) the infantile type, and (4) the septicemic type. It may run an acute course, the patient either dying or recovering after the use of serum; it may be greatly prolonged so that many doses of serum need be given before recovery or death ensues; it may relapse after apparent recovery, indicating perhaps some walled off focus of infection; and there is a class in which blocking occurs at the base of the brain which is very apt to be fatal.

The symptoms of the average case are familiar to all. The onset is acute, frequently with a chill. There are fever and headache. The patient is irritable, restless, and vomits. Shortly, the typical signs of meningitis develop, notably hypersensitiveness, delirium, stupor, convulsions, rigidity of the neck, increased reflexes, irregularities of respiration, Kernig and Brudzinski signs.

The fulminating type is the most malignant form. It runs a very acute course and usually kills within a few hours to a day. The picture is that of a violent general sepsis that utterly overwhelms the patient. If these fulminating cases can be tided over the first forty-eight hours they may respond to treatment.

The infantile type is easily overlooked in its early stages, starting usually as an obscure fever with gastro-intestinal symptoms, followed by convulsions, strabismus, and bulging of the fontanel. The bulging fontanel is the most important finding.

The septicemic type has only recently been recognized. All fulminating cases with purpuric rashes are probably of this character. In addition there is another group with a prolonged febrile state, frequently arthritic symptoms, and a mild roseolar or petechial eruption, much resembling typhoid fever. These patients may develop meningeal findings later, though some never do and eventually recover.

The usual type of meningitis may become chronic. The symptoms of this condition are continued irregular temperature, decided emaciation, vomiting, hydrocephalic cry and convulsions. The cause of this picture is frequently a posterior basic meningitis with a collection of plastic exudate at the base sealing up the foramina and giving rise to a hydrocephalus.

Relapses occur at times, probably in 5 to 10 per cent of cases. Many of these are due to the fact that the patient is not entirely well at the time of his supposed recovery and the process flares up. Under active treatment they often clear up. The patient should not be discharged till the spinal fluid is normal and its culture negative.

Conjunctivitis accompanies the disease very frequently; the discharge may show meningococci. Herpes when present also may show the organism in smear. Irido-choroiditis, usually purulent, occurs at times.

Deafness is one of the common sequelæ. Hydrocephalus occasionally follows. Mental deficiency results much less often than would be expected.

THE ORGANISM

The causative organism of cerebro-spinal meningitis is the meningococcus, often spoken of as the diplococcus intracellularis of Weichselbaum. This organism is a quite typical gram-negative diplococcus. It is practically always found at some stage of the disease in the spinal fluid, and often in blood cultures. In the fluid it may be intra- or extracellular, the relation to the leukocytes depending to great extent upon the intensity of the disease and the response to treatment. In severe cases there are many organisms, mostly extracellular. In favorably responding or milder cases they are fewer and mainly intracellular. Culture of the fluid usually shows a growth in eighteen to twenty-four hours. The organisms are more likely to be found in the last portion of the fluid withdrawn. The results of a Gram stain are not always reliable. A definite diagnosis must never be made on the result of a stained smear; it must rest on the results of a culture.

Meningococci fall into two large biologic groups, the typical meningococcus (Group I) and the parameningococcus (Group II). A great variety of intermediate strains related to one or the other of the two large groups occur, however, and Gordon has classified these intermediates into two groups which he calls groups three and four. The biologic differences are of great importance, particularly in the identification of the organism and the produuction of a potent serum. The meningococci of groups one and two are responsible for seventy-five to eighty per cent of all cases of meningitis.

It is generally believed that the toxic sub-

stances derived from the meningococcus appear as a result of massive cell autolysis (endotoxin) though highly toxic filtrates may be obtained from cultures in which there has been comparatively little cell autolysis. Schwartzman has succeeded in producing skin reactions with filtrates of meningococcus cultures and in being able to neutralize them in a high percentage of cases with antimeningococcic serum.

The fluid withdrawn at the first puncture most often yields the organism. However, I have seen organisms appear *only after* the giving of the serum. The best culture medium for its growth is probably the dextrose semisolid agar as prepared by Gosling. In 89 consecutive meningococcic cases tested by her, growth was obtained in eighty-one; at times growth occurs in this medium only; frequently growth occurs here when a smear is negative for organisms. Growth occurs in from eighteen to ninety-six hours. The identity may be proven by agglutination and fermentative tests.

PATHOLOGY

The nose and throat are the portals of entry of the organism into the body. Meningitis is first a case of naso-pharyngitis. Invasion from the nose and throat may occur in two ways: (1) By direct infection from the nose through the lymphatics; (2) by a primary invasion of the bloodstream causing a meningococcus septicemia with secondary localization in the meninges. Most authorities now favor the latter course.

It is probable that there is a brief invasion of the meningococcus in the blood in practically all cases of meningitis. Herrick found positive blood cultures in 45 per cent of his cases. Other authors give the incidence as 25 per cent. The duration of the blood infection before the development of meningitis may be extremely short; the two may appear coincident, as in the fulminant cases; or it may be of days' duration. It is possible to have a septicemia without infection of the meninges. This is usually transient, perhaps not over a few hours, but on the other hand it may last for days or even months, at times to be followed by an acute meningeal invasion. Some cases never pass beyond the septicemic stage. Richmond reports twenty-one cases of which seven remained septicemic only. Even fulminating cases ending fatally may show no evidence of meningitis at necropsy. Septicemic cases are less frequently seen in children.

Before the entry of the organism into the cerebrospinal fluid is described it will be wise to describe the circulation of the fluid. The fluid is secreted principally by the choroidal plexuses in the lateral ventricles. It flows through the foramina of Monro from the lateral ventricles into the third ventricle, through the aqueduct of Sylvius into the fourth ventricle, and from here gains access to the cisterna magna and the basilar system of cisterns by way of the foramina of Luschka and Magendie. The cisterna magna may be regarded as the distributing center of the fluid, as from here it gains access to the other cisterns and to the cerebral and spinal subarachnoid spaces.

Absorption of the cerebrospinal fluid occurs through the arachnoid villi into the cerebral sinuses or into the veins of the meninges.

Present opinion points to the entrance of the organism into the central nervous system via the blood stream through the choroidal plexuses. To do this the usually efficient barrier of the plexus must break down. Many observers contend that the cortex is also involved primarily, along with the lining of the ventricles. Leukowicz believes we must consider meningitis as a primary ventriculitis (choroido-ependymitis) while the lesions of the meninges constitute only a secondary phenomenon. He believes that no autonomous meningitis can exist independently of the ventricular infection nor require a serum treatment on its own account. Rolleston and Dopter agree with this in part. The barrier of the choroid plexus is overcome through inflammation of the plexus itself, much as in the metastatic skin lesions before mentioned, weakening the epithelial lining of the plexus villi and allowing the organisms to pass through.

If the consistency of the brain is soft and flaccid, as exists in infants, the cerebral edema and condition of pressure soon results in an obliteration of the sulci of the cerebral convexity. In those parts of the subarachnoid spaces that have thus lost their connection with the ventricles the inflammatory processes disappear quickly, according to Leukowicz. If large purulent layers are already present in the sulci the pus disintegrates, young connective tissue grows in and forms sclerotic plaques. The sulci of the convexity being closed, removing the largest area

of fluid absorption, the cisternæ of the base remain the only part of the subarachnoid space within the cranium communicating with the ventricles. Consequently these alone remain filled with pus. This type of block explains the pathogenesis of the posterior basic type of meningitis as seen in infants and its resultant hydrocephalus. Adhesions obliterating the basilar foramina can also bring about this picture.

With the progress of age, however, the consistency of the brain becomes more firm and resistant, and changes of configuration leading to obliteration of the sulci can develop only after long duration. The subarachnoid spaces of the convexity thus remaining passable for a long time, are the site of an active resorption of the fluid and also of an abundant accumulation of its cellular elements. As the drainage of the ventricles is sufficient, hydrocephalus does not soon develop. On the other hand, the brain, not being able to markedly change its configuration, suffers from the pressure exercised on it by the osseous cranial walls and manifests this by violent cerebral symptoms.

Another spinal type of block may occur, due to an edema of the cord. This spinal type is more common in infants and is related to the increased size of the cord in proportion to the size of the spinal canal at this age. Occasionally a condition of "dry canal" may simulate spinal or basilar block and must be differentiated from them.

DIAGNOSIS

The diagnosis of cerebrospinal meningitis rests upon the finding of the meningococcus in the spinal fluid. The fluid when taken early in the septicemic invasion stage may be perfectly clear and normal microscopically. However, several hours later, turbidity may be manifest. The usual meningococcic fluid is frankly turbid. As a rule there is a decidedly increased cell count running into hundreds or thousands per cubic millimeter. The cells are usually polymorphonuclear in type, though occasionally lymphocytes predominate. Lymphocytes are apt to predominate in the very early fluids. There is an increased amount of protein and a decreased amount of sugar present. The gram-negative diplococci are almost always to be found in varying numbers on smear, and grow fairly readily in suitable culture media.

The white blood count is of varying significance. There is practically always at least a moderate leukocytosis. In some epidemics, however, it is greatly increased, with a high percentage of polymorphonuclears. Richmond reports counts from twenty thousand to fifty thousand with 90 per cent polymorphonuclears. It is safe to state that in an unexplained fever with a white blood count of over twenty thousand, meningitis should be suspected.

The meningococcus may be found in blood culture. Special media such as ascitic broth, blood broth, or blood agar, should be used, as the organisms grow poorly in ordinary media. Blood cultures may be very important in the recognition and treatment of meningococcic sepsis.

THE SERUM

Before treatment is discussed a few words about antimeningococcic serum are in order. The serum is produced by the injection of horses with living or dead cultures of the meningococcus over a period of time, and systematic bleeding thereafter. All serums are supposedly polyvalent for the four different strains of the Gordon classification as specified by the Government. The serum of some laboratories contains antibodies for additional strains. The problem of determining the best method of preparing a serum of value against all strains of the meningococcus still is incompletely solved. It is possible that a serum prepared with the use of only the four Gordon strains may be more potent than one prepared with a greater number. It has been shown that the concentrations of the different types of antibodies varies with the period after injection that the horses are bled. The value of therapeutic serums may be tested by the use of several immunity reactions, of which the agglutination test seems to be the best. It, however, is not a consistently accurate measure of the serum's real therapeutic value.

The serum when prepared contains agglutinins, complement-binding antibodies, bacteriotropins or opsonins, and anti-endotoxins. Its effect is to injure the meningococcus, reduce it in numbers, alter its size and staining properties and render it more readily taken up by the leukocytes (phagocytosis). The disintegration products of the organisms are also neutralized by the anti-endotoxins of the serum.

TREATMENT

The symptom-complex of antimeningitis consists of symptoms of sepsis from the purulent

inflammation, symptoms of meningeal irritation, and pressure symptoms from the collection of exudate in the ventricles and subarachnoid space. In treatment, sepsis must be combated by serum injection into the meninges, and the equally important hydrocephalic pressure symptoms relieved by withdrawal of spinal fluid. It may be accepted as a general rule that unless the serum is introduced directly into the meninges the course of the local conditions will be little affected. When blood stream infection is present, or presumably so, subcutaneous or intravenous serum is also to be used.

Treatment may be divided into two parts: (1) treatment with serum; (2) general management.

If we grant that the original site and "feeder" of the infection is in the ventricles, the logical aim is to provide a sufficiently intense serum reaction in the ventricles and to maintain it for several days till disappearance of the meningococci occurs. This does not mean that serum necessarily must always be given directly into the ventricle itself. Serum injected intraspinally by the lumbar route penetrates into the ventricles in quite a sufficient concentration in almost 80 per cent of cases. The concentration of serum in the ventricles shortly after lumbar injection is often as high as 10 per cent and even after twenty-four hours may still be from 1 to 3 per cent. Serum injected into the general circulation is secreted into the spinal fluid only in small quantities and very slowly so that its action in the central nervous system cannot be depended upon. All cerebrospinal meningitis, therefore, demands intraspinal treatment. When meningitis is suspected, lumbar puncture should be done at once. The old dictum "a cloudy fluid demands antimeningococcic serum until *definitely proven* other than meningococcic meningitis" still holds true. Failure to observe this rule is often costly, as treatment with serum is most likely to give results when given early in the disease, the earlier the better. No harm, other than obscuring the picture for a time, results from the giving of serum, if proper precautions are taken. Meningococci occasionally are not found in cloudy fluids for several days, during which precious time is lost. The mere giving of serum may serve to bring the organisms forth. Serum should be continued until the organism found is proven beyond question not to be the meningococcus.

Specific bacterial immune bodies, agglutinins, opsonins, and antibacterial substances are usually absent, or present only in very small quantities, in the cerebrospinal fluid in meningococcic meningitis. Immune substances of similar type have been demonstrated in the blood of meningitis patients, however.

When spinal puncture is performed enough fluid should be withdrawn to exceed by five cubic centimeters or more the amount of serum it is desired to give. This is a safe working rule. The serum given should be allowed to run in slowly by the gravity method. Neal has at times given more serum than the amount of fluid withdrawn when it has run in easily by the gravity method, carefully observing the patient during the procedure. This is not to be recommended to the average practitioner.

The amount of serum to be given at each intraspinal injection depends upon the age of the patient. Young infants may be given 10 to 20 c.c.; adults up to 60 c.c. The interval between the injections has varied in different hands from eight to twenty-four hours. As serum injected intraspinally does not entirely pass out of the subarachnoid space for twenty-four hours, a twenty-four hour interval seems logical. Neal has thought it probable that removing fluid at more frequent intervals might remove cells that had been stimulated to a high degree of phagocytic power and were therefore actively combating the infection. Since the serum has so little bactericidal power itself, the administration of large amounts at frequent intervals seems unnecessary to her, besides subjecting the patient to considerable added pain and discomfort. Sophian advises a twenty-four hour interval. However, when the increase of pressure is great, lumbar puncture for its relief may be done at more frequent intervals and serum given. In Neal's hands the twenty-four hour interval of injection has yielded a mortality of 23 per cent in a large series of patients, a mortality rate lower than the average.

The average case requires from four to eight injections although at times a good many more are necessary. There are two main guides to the continuation of treatment: (1) the findings in the fluid at each puncture; (2) the clinical condition of the patient. While the fluid has a tendency to become clearer macroscopically as the disease improves, it may remain quite turbid

due to the excitation of leukocytosis by the serum itself. As the patient responds, the organisms in the fluid become less numerous and more frequently intracellular, frequently appearing clumped together and failing to grow in culture. Such findings in conjunction with clinical improvement of the patient are indications for cutting down on the frequency of injections to 36 to 48 hours. Serum treatment should be continued until spinal fluid smears are negative for bacteria and cultures from it show no growth, always being prepared to institute treatment again if the patient's condition becomes worse or the fluid findings retrogress. The spinal fluid cell count is not an accurate guide to the discontinuance of serum. It is frequently necessary to do one or more spinal punctures after serum is discontinued for the relief of pressure symptoms.

Reaction of spinal fluid injection.—The injection of serum is usually accompanied by pain referred to any or all of the region affected. It usually subsides by the end of the operation, though it may continue for several hours. Usually there is a drop in blood pressure, at times quite marked. Fluid may be withdrawn till the pressure is normal, which roughly corresponds to ten to twenty drops per minute. Shock occurring during or after injection is usually due to the injection of too large a quantity of serum, too rapid injection, or undue pressure excited during its injection, and at times calls for the immediate withdrawal of serum and fluid when possible. Adrenalin and atropin may be used.

Intravenous serum. — The general opinion seems to be that intravenous serum should be given only to those cases with evident sepsis as manifested by joint signs, rash, or positive blood cultures, or in the very early cases when the patient is presumably in the septic stage. It may also be given when no improvement occurs after thorough spinal and cisternal treatment. Herrick advised 80 to 150 c.c. per dose; other authors use 20 to 50 c.c. The dose may be repeated at twenty-four hour intervals for several days until improvement occurs. It must be remembered that intravenous serum is prone to elicit severe and even fatal reactions; this fact has led several authors to discontinue its use. Attempt at desensitization should always precede its administration. Serum may be given by the subcutaneous or intramuscular route with greater safety.

Cisternal treatment.—When there is clear indication of block, puncture of the cisterna magna and serum injection at this site is often of the greatest aid. It also may be used when treatment via the lumbar route is not giving results. I am not going to attempt to describe its technic; suffice it to say that it is not overly difficult. Its advantage lies in the fact that serum reaches the strategic points more surely than by the lumbar route. As a rule cisternal treatments are well tolerated by patients. The dangers of cisternal treatment are intracranial compression, a jamming herniation by release of pressure, hemorrhage over the medulla from trauma to the spinal veins, and trauma to the medulla or cerebellum from improper puncture. Such occurrences are rare with correct technic. It is well to remember that patients who show evidence of anaphylaxis do not respond well to intracisternal serum, possibly as a result of an edema of the brain.

Ventricular treatment.—Some feel that the ventricular injection of serum is superior to the cisternal injection, believing it less dangerous and even more efficacious. Particularly is this felt in the case of infants, where its performance is usually a rather simple matter. Use of the cisternal or ventricular routes, when indicated, should result in further lowering of the mortality.

Combined treatment.—Some authors routinely treat cases by a combination of lumbar and cisternal or ventricular injections with good results.

General care.—The general care of the patient must not be lost sight of. Sedatives are necessary for restlessness, elimination must be secured, the fluid and food requirements met. If necessary, tube feeding should be used. Saline subcutaneously may be administered. At times blood transfusion is valuable.

The patient should not be discharged until the spinal fluid and cultures are normal and he has been temperature-free for at least one week.

Other types of treatment.—The treatment of meningococcic sepsis without meningitis is intravenous serum. Richmond treats these cases by intraspinal injection as well and feels that it sometimes prevents meningeal infection.

Neal, Banzhaf, and their co-workers have used an antibody preparation made by concentrating the serum, partially purifying its antibodies by precipitation, and then redissolving in sodium chloride. The agglutinating power of this antibody preparation is much higher than

the serum from which it is made. It is given intraspinally in doses of 20 c.c. every twenty-four hours. With such a satisfactory preparation their mortality in twenty-four cases was 12.5 per cent. Neal tells me in a personal communication that this procedure is still in an experimental stage.

An autogenous vaccine has been used in chronic cases both subcutaneously and intraspinally, with good results at times. The use of convalescent serum is barely mentioned in the recent literature and the value of this treatment I do not know.

PROGNOSIS

The earlier the diagnosis and institution of treatment, the better the prognosis. The spinal fluid sugar findings offer distinct prognostic aid. The normal fluid sugar content is forty-five to sixty-five milligrams per hundred cubic centimeters. Before treatment is instituted it drops as a rule to about twenty milligrams but with improvement it gradually rises until it becomes normal again about the ninth day. Both the fermentative action of the meningococcus and the glycolytic action of the polymorphonuclear leukocytes have been considered the etiological factors in the decrease of the spinal fluid sugar. In the fatal cases it remains low. In infants it is often as low as thirteen milligrams and increases much more slowly, not reaching normal during treatment. Caffey and his co-workers believe the slow return to normal in infants is due to the slower subsidence of the inflammatory cellular reaction which they exhibit.

Estimation of the spinal fluid chlorides does not give valuable prognostic information.

Change of serum.—When the patient does not show the expected improvement in spite of thorough treatment it is often wise to use a different serum. It has several times been shown that some brands of serum will not control certain epidemics. The agglutination test as a measure of a serum's effectiveness is not certain; one must depend on the therapeutic response in determining the value of the serum used.

Serum sickness occurs in about 70 per cent of all cases, usually from the fourth to the fifteenth day.

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DIFFUSE SUBCORTICAL SCLEROSIS OF THE BRAIN*

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THE confusion of classification and terminology which surrounds the concept of diffuse cerebral sclerosis and the lack of sufficient fundamental data with which to clarify our concepts justifies the reporting of further individual cases of the condition. During the time in which the number of reported cases is small, the publication of additional cases will likely increase this confusion; but at least it will tend to break down insufficiently grounded nosological hypotheses, though it may leave little in the way of satisfactory understanding in their place. Nevertheless, we shall probably eventually acquire through such reports a sufficiently large series of cases so that the more obvious features of the disease can be seen with reasonable perspective, whereupon our terminology can be constructed with sufficient logical basis, such that some general acceptance of terms may be attained.

Coinage of names for the condition and its types has been indulged in by various writers to such a point that one becomes quickly disoriented in the maze of words one might be expected to keep in orderly array for the condition and its variants. For example, the following terms have all been proposed as the most suitable for one or another phase of the condition or for the condition itself:

1. *Encephalitis periaxialis diffusa* or Schilder's disease. The term was introduced by Schilder[1] in 1912 to designate a type of diffuse cerebral sclerosis involving the white matter of the hemispheres in childhood and separated from the diffuse gliomas of the brain. He assumed a toxic etiology which justified the implication of brain inflammation in his terminology. Bielschowsky[2] limits the term to such cases of cerebral sclerosis as show strong evidences clinically or pathologically of an inflammatory process. He presents such a case. Other writers (for instance Bouman[3] and Collier and Greenfield[4] and several Americans) have applied the term to cases which they considered primary degenerative. Ferraro[5] presents his cases as the "familial form of encephalitis periaxialis diffusa"; thus hereditary forms have also been classified under Schilder's terminology.

2. *Progressive degenerative subcortical encephalopathy.* This term was suggested by Globus and Strauss[6] to supplant Schilder's term of encephalitis periaxialis diffusa. Globus and Strauss obviously considered the condition as primary degenerative.

3. *Symmetrical centro-lobar cerebral sclerosis* (La sclérose cérébrale centro-lobaire a tendance symétrique) is the term used by Foix and Marie[7] to mean a chronic form of encephalitis periaxialis diffusa of Schilder, or a form which may come to spontaneous arrest.

4. The term *aplasia axialis extracorticalis congenita* (Pelizaeus-Merzbacher disease) is properly applied, according to Liebers,[8] only to cases occurring in a single family which has been reported through four generations by Pelizaeus, Merzbacher, Spielmeyer and Liebers. The condition occurred in fourteen members of the family and in each case ran a chronic course after an onset in infancy.

5. Krabbe[9] described a *familial infantile form of diffuse brain sclerosis* with an acute course.

6. For the hereditary forms in general of primary degenerative diffuse sclerosis involving the subcortical white matter, Bielschowsky and Henneberg[10] have introduced the designation *leucodystrophia cerebri progressiva hereditaria.*

It is hoped that the present report will not add to the confusion.

PRESENTATION OF A CASE

A white boy, aged 2.5 years, was admitted to the Pediatrics Service of the University Hospital November 11, 1929. His birth was normal. He weighed eight pounds at birth. He was breast fed for nine months. Child was perfectly well until July, 1929, at which time he weighed twenty-six pounds. Since that time the weight has been stationary. Sat up at 8 to 9 months; walked at 1.5 years; had his first tooth at 17 months; had sixteen teeth at the time of admission. He began talking at one year but has never talked very much. Since the onset in July, 1929, the patient has ceased speaking altogether. It was noticed by the parents that the baby fell down frequently without stumbling; later was entirely unable to maintain his balance. Shortly thereafter he became unable to walk at all,

*From the Division of Nervous and Mental Diseases, University of Minnesota Medical School, Minneapolis. Presented before the Minnesota Academy of Medicine, October 8, 1930.

and in August he could not crawl. At that time stiffness of both legs was noticed, and a short time later he could sit only with difficulty, being obliged to double his legs under him (squatting) to maintain his trunk in the erect position. Since early in October he has been unable to use his hands and arms. Both extremities are kept extended and no voluntary movements are made. The head has been held in opisthotonos for some weeks.

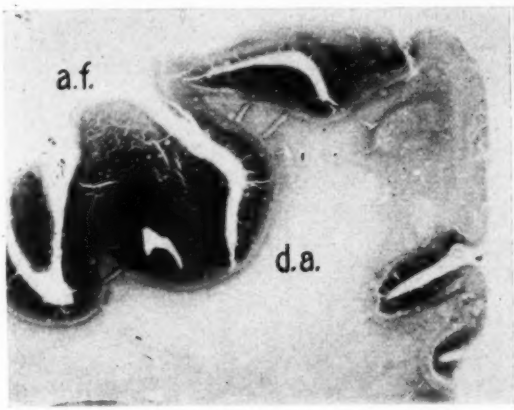

Fig. 1. Left frontal area of brain. a.f.—arcuate fibers. d.a.—degenerated area, the subcortical white matter. Weigert's myelin sheath stain. X 2.

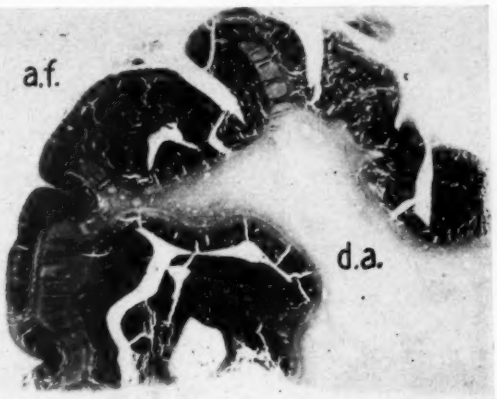

Fig. 2. Left parietal area. a.f.—arcuate fibers. d.a.—degenerated area, the subcortical white matter. Weigert's myelin sheath stain. X 2.

There was no previous illness. Father and mother are both living and well. They are cousins. The mother had an operation for goiter. The patient is the second child.

A brother lived to the age of 2 years 9 months and became ill much like the patient, developing speechlessness. He was never able to walk and became spastic in all extremities before death and died finally of bronchopneumonia.

Examination.—Physical examination by the pediatricians showed fair development but slight emaciation. The pupils were fixed to light and dilated. The eyegrounds were negative. Throat and mouth showed no pathology. Heart, lungs and abdomen were normal. All the extremities showed atrophy of the muscles. The reflexes were increased in the upper extremities. Reflexes could not be obtained in the lower extremities because of spasticity. Both lower extremities were very spastic and held in constant extension.

Laboratory findings.—Blood examination showed hemoglobin 91 per cent, red blood cells 4,910,000, white blood cells 11,000, polymorphonuclears 40, lymphocytes 58, monocytes 1, eosinophiles 1 per cent. Spinal fluid: normal pressure, Wassermann negative, colloidal gold curve negative. Urinalysis showed a few red blood cells and a few white blood cells; otherwise the urine was normal. As the patient was admitted for diagnosis only, no therapy was attempted.

The writer examined the child neurologically November 14, 1929. The examination was unsatisfactory. The child cried perpetually and could not coöperate in the least. He appeared completely demented. He lay in opisthotonos with the legs extended and arms abducted. The pupils were completely dilated as though by homatropine. They were fixed to light. No facial paralysis was present. The cranial nerves were otherwise not satisfactorily examined. The reflexes were not obtainable because of the marked spasticity in all extremities. He moved the arms very little though he could grip with his fingers. The legs were moved freely but were extremely spastic, with adductor spasm. The toes were continually in the position of a positive Babinski. Clonus could be elicited in the right foot. The condition was thought to be probably diffuse cerebral sclerosis. The patient died December 17, 1929.

Postmortem examination.—This was performed about twelve hours after death. The body was that of a fairly well developed, rather poorly nourished baby boy, 88 cm. long and weighing about thirty pounds. Rigor was very marked; purplish hypostasis was present posteriorly. Slight cyanosis was present. There was no edema or jaundice.

The peritoneal cavity was normal. The appendix was free and anterior. The pleural cavities were normal. The pericardial sac was normal; it contained no excess of fluid. The heart weighed 60 grams. All the chambers and valves were normal. There was no anomaly of the vessels. The root of the aorta was normal. The thymus weighed 20 grams and was normal grossly on the surface and on section. The left lung weighed 170 grams, the right 200 grams. Both were nodular and congested throughout and pus could be be expressed from both on section. The spleen weighed 40 grams and was normal grossly. The liver weighed 360 grams and was slightly pale; otherwise entirely normal. The gallbladder was normal. The gastrointestinal tract, pancreas and adrenals were normal. Each kidney weighed 40 grams and there was slight cloudy swelling. The bladder and genital organs were normal. The aorta and lymph nodes were normal.

At autopsy no abnormalities of the brain were noticed. The brain was cut into coronal sections at the time and was delivered in this condition to the neuropathology

laboratory. Here no gross abnormalities of the surface of the brain were seen. Palpation of the brain substance showed a marked increase of consistency, especially in the white matter underneath the cortex. This increased consistency was not uniformly diffuse but could be felt more under certain convolutions than

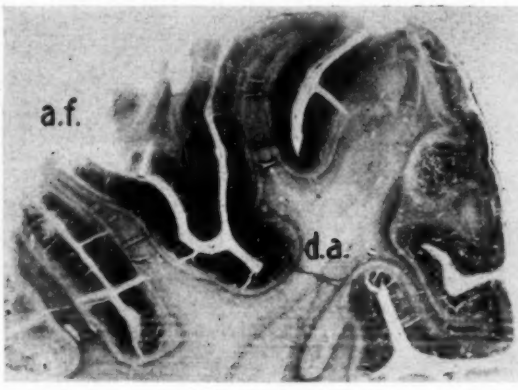

Fig. 3. Left occipital area. a.f.—arcuate fibers. d.a.—degenerated area, the subcortical white matter. Weigert's myelin sheath stain. X 2.

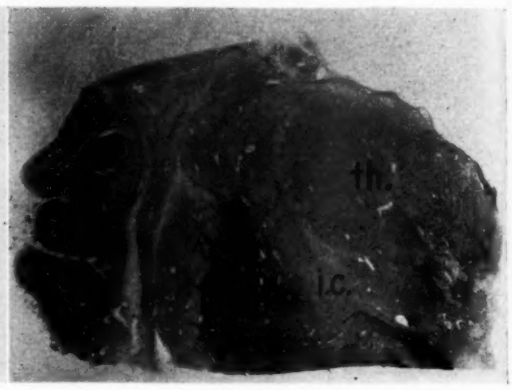

Fig. 4. Right basal ganglia. gl.p.—globus pallidus. i.c.—internal capsule. p.—putamen. th.—thalamus. Weigert's myelin sheath stain. X 2.

under others, though there was no tendency for the firmness to be more marked in one or another lobe of the brain. Sectioning the brain with a razor produced the sensation of cutting a sclerotic organ as compared with normal brain.

Microscopic findings.—The accompanying figures illustrate fairly well the essential features of the microscopic pathology. Figures 1 to 4 show Weigert sections of various parts of the cerebral cortex and the basal ganglia. It appears at this low magnification that the myelin sheaths of the centrum semiovale are practically absent. Immediately beneath the gray matter of the cortex is a fine strip of remaining undegenerated myelin sheath fibers following the topography of the base of the cortex. These are arcuate or U fibers of the cortex running from convolution to convolution. It has often been stated that the arcuate fibers are not degenerated in Schilder's disease. However, one sees with the high power of the microscope that not only are the arcuate fibers not degenerated in this area but also the fibers running vertically between the cortex and the centrum semiovale. Thus the preservation is evidently not a system preservation but a regional one with which we are dealing. In Figure 4 the degeneration can be seen extending down into the internal capsule. The fibers of the globus pallidus and putamen are rather markedly degenerated. The fibers of the thalamus appear to be less numerous than is normal. Under the high power the subcortical white matter is seen to be extremely degenerated as regards myelin sheaths with the exception of the arcuate fibers. Only a scattered number of them remains (Figs. 5 and 6). With Bielschowsky's stain for nerve cell processes it is seen that about as many axis cylinders persist as there are myelin sheaths (Fig. 7). Sections of the pons, medulla and spinal cord (Fig. 8 and 9) are of no particular interest excepting that the pyramidal tracts are degenerated as an apparent secondary degeneration and in the medulla the myelin sheaths in general appear to be somewhat scanty.

Cellular stains of the cortex show that the cells of the superficial layers are about as numerous as one ordinarily finds and are well preserved (Fig. 10). The cells in the 6th and in the deeper parts of the 5th cortical layers are less numerous than normally and are undergoing definite degeneration as shown by chromatolysis, fragmentation and nuclear abnormalities. The cells of the globus pallidus are distinctly degenerating and less numerous than normally. In the putamen the cells appear to be about normal. In the thalamus they are apparently moderately decreased in number. The cells of the third nerve nucleus and of the substantia nigra are likewise moderately decreased in number and show degenerative changes. In the medulla the cells are normal except for those of the inferior olive, which are rather strikingly reduced in number; those remaining show only mild evidences of any degenerative process. In the spinal cord the nerve cells appears to be about normal.

Hematoxylin and eosin sections of the white matter beneath the cortex (Fig. 11) show a rather marked increase in the number of glial cells in the region. A large proportion of these glial cells is made up of protoplasmic glia. These cells constitute an unusual picture in that the markedly increased cytoplasm appears homogeneous except at the periphery of the cells, where it is extremely grandular. The granules stain deep red with eosin. Some of these cells appear to have undergone a partial disintegration. Staining by the various silver carbonate methods to bring out the different types of glial cells shows that the vast majority of the glia is made up of astrocytes. The astrocytes have increased in great number and their processes, running in every direction, make a heavy network of coarse glial fibers. It is undoubtedly this increase in astrocytes and their fibers which is responsible for the increased consistency of the white matter of the brain (Figs. 12 and 13). The

increase of astrocytes is most marked in the region of the cortical arcuate fibers which have not yet undergone extreme degeneration.

Various parts of the brain were stained with Sudan III to demonstrate fat. This can be seen piled up in scavenger cells along blood vessels in scattered areas.

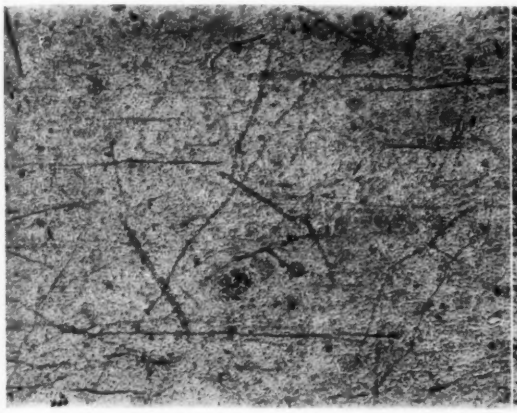

Fig. 5. White matter of left occipital region where myelin sheaths are very sparse. Weigert's myelin sheath stain. Magnification about X 150.

Fig. 6. White matter of left occipital region in the area of the arcuate fibers. Weigert's myelin sheath stain. Magnification about X-150.

It can be found in glial cells in small amounts though it is sometimes collected in large amounts in the large protoplasmic glial cells (Fig. 14). Fat-containing glial cells are more abundant in the region of the cortical arcuate fibers than elsewhere in the white matter. Increase of astrocytes in the basal ganglia and likewise in the midbrain is a distinct part of the picture microscopically.

The vessels of the brain show only mild abnormalities. These are evidenced by the collection of scavenger cells in the adventitial spaces along with small numbers of perivascular lymphocytes. The vessels are not thickened and there are no evidences of thromboses. The meninges appear normal throughout.

RECENT CASES FROM THE LITERATURE

Bouman,[3] Collier and Greenfield,[4] Stewart, Greenfield and Blandy,[11] and Globus and Strauss,[6] have reviewed the early literature of Schilder's disease so that it is unnecessary at this point to go over these early cases. These authors together have reported and reviewed about 48 cases. In the present paper I shall review 15 cases that have appeared subsequently to the articles by these writers, or cases which they did not include in their reviews. Together with the personal case reported by me, the total number of pathologically verified cases of diffuse subcortical sclerosis is thus 64. Parts of the following reports are taken verbatim from the former writers. Only those used entire are enclosed in quotation marks.

No. 1. Schilder,[12] 1924. Female, age 37. One month after an attack of grippe, the patient lost her sense of smell on the left side. Incontinence of urine appeared and the patient developed headaches. She became markedly constipated. The menses of the fifth month after onset were premature. First examination five months after the onset of the trouble. This showed an objective left-sided decrease of smell with negative findings in the nose. Apraxia of the eyelids and to a less degree in the upper region of the face was present. There was a slight right-sided hemiparesis of the pyramidal type. An uncharacteristic tremor of the hand was present, more pronounced on the right side than on the left. The patient tended to fall backwards in a retropulsive fashion, which carried with it, however, a certain apparent voluntary character. Bilateral beginning choked disk was present. Psychically the patient was apathetic, dull and showed a lack of initiative and interest. She was incontinent. A presumptive diagnosis of brain tumor was made and the patient was operated upon. She died about eight months after the onset.

No. 2. Scholz,[13] 1925. This writer has reported the clinical findings of five cases out of a single familial group. Only his second case has been described pathologically. Consequently, it is the only one which will here be summarized.

The hereditary features of Scholz's cases are so complicated that they will not be repeated here. The patient developed normally until the age of eight years, when he became ill with a severe headache and vertigo, with decrease of his mental activity, slowing of speech and spastic paralysis of the extremities, together with a slight sphincter involvement. At the same time he had only a slight decrease of his visual acuity with a correspondingly slight pallor of the temporal halves of the optic disks. Examination early in the disease showed a spastic paralysis of the extremities, slight cranial nerve symptoms and negative spinal fluid. Multiple sclerosis was considered most likely. A half year later he had

a total right-sided spastic paralysis with beginning contracture of the fingers and a high grade spastic paralysis of the left side of the body. He also had very evident paralytic phenomena in the cranial nerves and marked disturbances of the sense of touch. Vision had grown much worse; speech was slow and painstaking. Three months later he was completely helpless in bed; shortly thereafter could not speak at all and chewing and swallowing became very poorly performed. He developed severe spastic contractures. One year after the onset he developed attacks of muscle spasm in the extremities associated with crying, apparently because of pain with the spasms. The patient was gradually becoming demented. About three years after the onset he first showed generalized clonic convulsions with loss of consciousness and vomiting over several days at a time with each attack. After four years he no longer reacted to auditory stimuli and gradually became weaker. Was first seen in Scholz's clinic in the second half of the fifth year of the disease. Was now completely demented; reacted only to severely painful stimuli with crying and with indications of flight and protective movements. Neurologically, as concomitant signs of the spastic paralysis, there were increased reflexes, clonus, pathological great toe phenomena. It was noted that on the right a partial, and on the left a total optic atrophy was present. Spontaneous chewing and sucking movements were present and could be brought out by touching parts of the mouth. Swallowing was purely reflex only after touching the soft palate. Also in Scholz's clinic there were noticed painful spasms of the extremities. April 21, 1922, fifteen days after coming into the clinic, he died with bronchopneumonia after an onset four years and ten months previously.

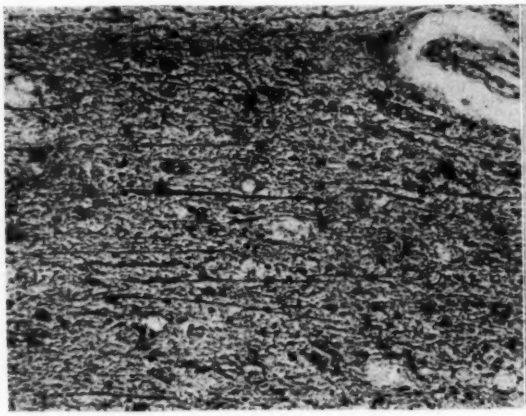

Fig. 7. Deep in the white matter of the right parietal region where myelin sheaths are sparse. For comparison with Figure 5. Bielschowsky's neurofibrillar stain. Magnification about X 150.

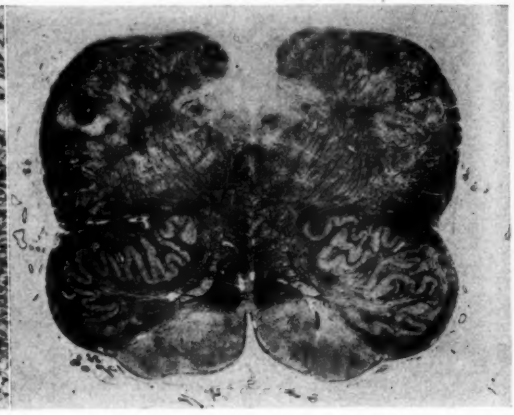

Fig. 8. Medulla oblongata. The pyramidal tracts are markedly degenerated. Weigert's myelin sheath stain. X 4½.

No. 3. Kraus and Weil,[14, 15] 1926 and 1928. Case was that of a Hebrew tailor, born in 1877, whose family and past history were unimportant. In 1911 while walking in the street, the patient suddenly fell to the ground helpless and could not get to his feet again without assistance. There were no convulsions and no loss of consciousness. Sensory disturbances, ataxia, and anesthesia to touch were noted in all four extremities, but disappeared after a few weeks. The cranial nerves were normal with the exception of slight deviation of the protruded tongue to the right. In December, 1911, there was slight concentric contraction of the visual fields. At this time he also showed: diminished sense of smell; a slight tremor of the hands; ankle clonus which was persistent on the right, exhaustible on the left; persistent knee clonus on both sides; the upper abdominal reflexes were present, the lower were absent on the left; a pseudo-Babinski sign on the right and markedly exaggerated knee jerks; no ataxia; occasional incontinence of urine. Condition remained unchanged for five years. He was re-examined in 1921, at which time the same neurologic status was revealed, with an increase of the eye disturbances, insufficient convergence of the right eye, weakness of the rectus internus and externus on both sides, irregular pupils with reaction to light but none to accommodation. The right arm was weaker than the left and the biceps and triceps reflexes were lively on both sides. There was slight ataxia of the left arm, marked ataxia of the right, and ataxia of both lower extremities with loss of sense of position of the toes. A marked Romberg sign was present. There were no sensory changes. The gait was peculiar; it required a cane and was characterized by small steps and shuffling of the feet. Every time the foot touched the floor there was hypertension at the knee. Speech was very rapid and scanning with a peculiar repetition of the last word of each sentence. The condition was progressive and in July, 1923, the patient could not stand or walk. There was a mild degree of dementia. In March, 1924, complete left hemianesthesia was noted. In June, 1924, the patient died after an acute attack producing left-sided convulsions and anesthesia.

No. 4. Ferraro,[5] 1927. Case 1. Woman. Age at

death in 1925, 32. Onset in 1913 at the age of 20, following a severe burn. Behavior has been queer since then. She thinks someone has cast a spell over her. She refuses to wash or bathe herself or to comb her hair. She is forgetful, leaves the change on the counter, talks in her sleep and to herself; is a spiritualist. In 1919 it was noticed that she was somewhat intellectually and emotionally deteriorated; knee jerks very active; pupils dilated, rather irregular, sluggish to light, especially on the right; decided Romberg present; tremor of the facial muscles and fingers; speech ataxic; gait shuffling; Babinskis negative; no sensory disturbances; mild leukocytosis of the spinal fluid; Wassermann on blood and spinal fluid repeatedly negative.

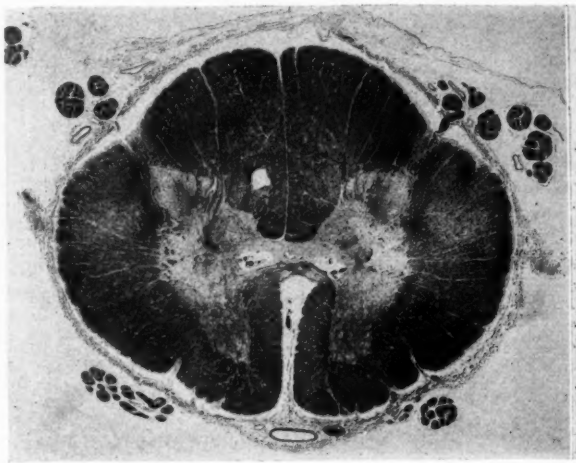

Fig. 9. Upper lumbar cord. Pyramidal tract degeneration. Weigert's myelin sheath stain. X 9.

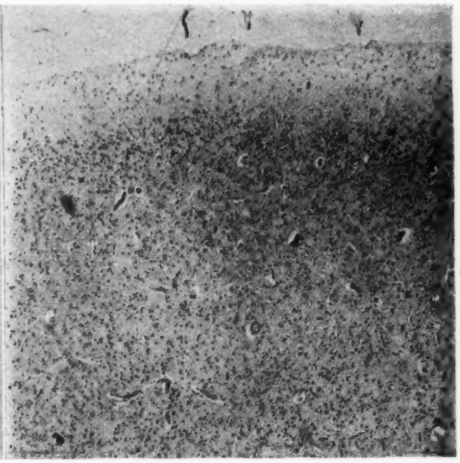

Fig. 10. Right parietal cortex. Thionin. Magnification about X 16.

The patient's parents were separated. Two brothers are said to have had "multiple sclerosis" (see the following two cases). Maternal grandmother insane, maternal cousin feebleminded. The patient was backward in school but social life normal. The neurologic symptoms progressively increased. In January, 1925, she was anemic and weak and had developed severe bed sores. Her speech was monotonous and sing-song. There were involuntary grimaces and distortions of facial muscles. Intention tremor was very marked. There was no nystagmus. She died in 1925 with a clinical diagnosis of multiple sclerosis.

No. 5. Ferraro,[5] 1927. Case 2. The patient was a brother of case 1. He was born in 1889. Except that he had never held a position for very long, his past history was essentially negative. He was drafted into the army in 1918. While at camp, about July 1, 1918, he began to be lame. He felt nervous at this time. Was discharged from the army as physically unfit in August, 1918.

While in the army camp he had begun to develop a paranoid trend. After leaving the army, he began to hear hissing noises in his ears and later had actual auditory hallucinations. He also began to have at this time explosive outbursts of laughter. He entered Bellview Hospital at his own request in September, 1919, claiming that he felt nervous, that he was very emotional and that his memory was not so good as formerly.

Examination showed the pupils somewhat irregular, but reaction to light was fairly prompt, though diminished in extent; the eyes were somewhat unsteady when he looked to the extreme right or left, but no clearly marked nystagmus was present. There was bitemporal pallor of the optic disks with contraction of the arteries and veins. His hearing was rather dull and he complained of a loud hissing in both ears. Cremasteric and abdominal reflexes were difficult to elicit though not entirely absent on either side. Babinskis negative. Muscular strength poor, but equal on both sides. The left side of the face was slightly flatter than the right, the tongue deviated slightly to the left, the gait was unsteady with the feet occasionally scraping the floor when walking, especially on the left side. All the muscular movements were clumsily performed, though the sense of position was not lost. Romberg sign negative. The fingers, tongue and lips were all fairly steady. No definite speech defect. Blood and spinal fluid Wassermann negative. Globulin ++ in the spinal fluid. The first spinal fluid test showed one cell, the second showed five.

Mentally the patient was dull and indifferent. He said that he felt depressed at times, but qualified this by saying he felt cheerful even when he was sad. He often laughed in a silly manner which he could not control. He still had hallucinations, memory was good for both recent and past events. He seemed rather childish and his judgment was poor. He died March 30, 1924, of bronchopneumonia. The neurologic diagnosis was multiple sclerosis.

No. 6. Ferraro,[5] 1927. Case 3. The patient is the brother of the two first cases. He was born in 1884.

No serious abnormalities of his development were noted until nineteen years of age, when he ran away from home. He stayed away for two years, working about the country in various places without much progress. At the age of twenty-one he fell a distance of sixteen feet, landing on his back. A week later he

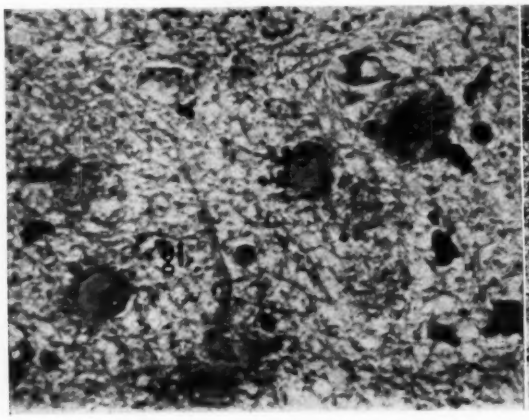

Fig. 11. White matter of right second frontal convolution. gl.—glial cell with much cytoplasm and peripherally arranged eosinophilic granules. Hematoxylin and eosin. X 300.

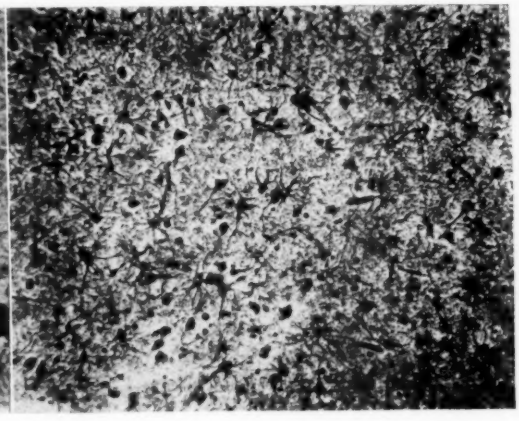

Fig. 12. White matter of left frontal region. Numerous glial astrocytes present. Hortega's silver carbonate stain for astrocytes. X 100.

had difficulty in walking. His foot appeared to be paralyzed and he had a staggering gait. He also became nervous and had spells of involuntary laughter. He was placed in a hospital, where he was somewhat hypochondriacal. He laughed without cause and explosively. The general examination was negative except for a slight dullness and prolonged expiration over the apex of the right lung. Urinalysis was negative. The tendon reflexes were all greatly increased. Ankle clonus present on both sides. Babinski phenomenon present on the right side. Superficial reflexes were absent. Station was good but the finger-to-finger and finger-to-nose tests were badly performed. His gait was stiff and awkward. The pupils were dilated and reacted to light and accommodation. He was unstable emotionally and seemed to be fluctuating between depression and exhilaration. On one or two occasions he threatened suicide. He was then paroled from the institution. In 1909 he developed a trophic ulcer on the left anterior tibial surface. In 1910 his health was fairly good and mentally he was in exactly the same condition as previously. The neurologic examination was the same as formerly except that the Babinski tests were positive on both sides, the gait was spastic in type, there was marked incoördination of the movements of the lower extremities, a coarse tremor was present which became more marked when fine movements were attempted. The speech had also become jerky in character and was termed scanning. In 1911 it was noted that he had become more unsteady in walking and occasionally fell. In 1912 he developed bowel and bladder incontinence. A spinal fluid test was done in May, 1914. The Wassermann, gold sol reaction and Nonne were negative. Cell count, 58 per cubic mm. Blood Wassermann, negative. He developed large bed sores and gradually became weaker until the time of his death, Oct. 26, 1916.

No. 7. Schaltenbrand,[18] 1927. "A girl, aged 14, came into the clinic because of headaches and pain in the right lower abdominal quadrant. The patient's mother has a depressive climacteric psychosis and the grandmother has suffered for years from a paranoid psychosis. On admission the patient was apathetic. She had a right facial weakness and hyperemia of the optic disk. During the next few weeks, she developed hypotonic right-sided hemiplegia, hypesthesia for touch and pain over the whole right side, right hyposmia, and left homonymous hemianopia. Shortly afterwards, a motor and later also a sensory aphasia appeared. The choking of the disks gradually increased during the next few weeks to from 4 to 5 diopters. Meanwhile the left leg became paralyzed, and all the paralyses slowly acquired a spastic character. The right arm went into flexion contraction. The head and eyes became strongly deviated, at first to the right and later to the left. Finally, the patient became entirely blind, aphasic and almost completely paralyzed, though in the left arm certain movements, athetoid in character, put in an appearance."

"One lumbar puncture gave negative results; another, a slight increase of globulin and 10 cells. The blood picture was normal, with the exception of a slight leukocytosis. The patient grew worse in spells which were accompanied by a subfebrile temperature. The disease lasted three months altogether."

No. 8. Bielschowsky,[3] 1927. The patient was born in 1868. His family history showed that his antecedents were a nervous type, otherwise normal. He, himself, is said to have had a tendency to fits of rage in the past. As a youth he had a middle ear inflammation with subsequent decrease in his hearing. Otherwise was well until the period 1918 to 1920, during which time without apparent cause all of his teeth fell out. In September, 1920, at the age of 52, he was sick for ten

days with fever, chills, joint pains and lassitude. Three weeks later he again had a febrile illness. He is said to have had yellow scleræ. The sickness was first considered malaria and then later an intestinal intoxication. There were no nervous symptoms. The patient has been impotent since this illness. In 1923, he was

Fig. 13. Same area as in Figure 12. The coarse ramifying fibers of the astrocytes make a dense network. X 400.

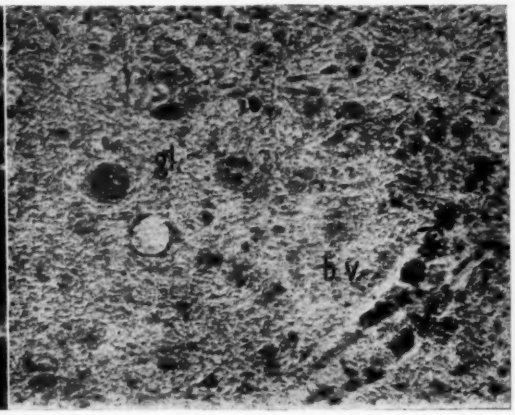

Fig. 14. White matter of frontal region. Sudan III stain for fat. gl.—protoplasmic glial cell filled with fatty granules. b.v.—blood vessel with a row of fat-filled scavenger cells in the perivascular space on either side of the vessel. X 300.

denied coverage for life insurance because his pupils had become unequal and reacted poorly. Later special investigation did not substantiate this finding. In June, 1924, he had another attack of febrile illness which lasted off and on until the first week of July. August 8, 1924, he was again taken sick, with confusion, weakness, inability to speak. The following day his temperature rose 1° to 2° C and he recovered on the fourth day. Examination on the fifth day was negative neurologically except for a positive Oppenheim reflex on the left. There was a hypalgesic zone on the left leg and a hyperalgesic zone at the nipple level running around the trunk. The patient swayed somewhat in turning abruptly on his feet even though his eyes were open. The visceral examination was essentially negative except that the liver edge could be palpated. Blood counts normal, no malaria, the Wassermann reaction normal. August 11 the sensory disturbance in the left lower extremity disappeared and the liver edge could no longer be palpated. September 1, 1924, he came home extremely tired and much agitated; the next day he complained of headache; the temperature was normal. September 3, he had an afternoon temperature of about 1° C. He complained of severe headaches, he was very difficult to awaken in the evening and was quite stupid. September 4, the temperature was 39.2° C. Slept most of the time, could be awakened, however; had a nearly complete motor and sensory aphasia. He could not even say his own name at times. When he did speak he used Spanish, though his mother tongue was German. An exact examination was impossible. He appeared not only temporally and spatially disorientated but also apractic. He was negativistic and incontinent. In the afternoon his sensorium was entirely clear. He had a slight paraphasia and perseveration. Examination was essentially negative, excepting that he had evident though not very marked spasms. The Babinski and Oppenheim signs were positive on the left and the abdominal reflexes were weaker left than right. He had a complete amnesia for the time of fever. September 5, he had slight spasms in all the extremities, especially in the adductors of the thigh, so that he had a cross-legged progression. He could take a few steps with help, but tended to fall backwards with the eyes open or closed. His gait was ataxic. There was no ataxia in the arms. He could not tell when his bladder was full and recognized that his water was passing only when the skin of his thigh became wet. September 8, felt perfectly well. Was examined eight days later by Professor Cassirer, who found absolutely nothing wrong with the patient. On September 25, after having meantime worked at his profession as an engineer, he came home complaining of weakness and appeared rather dull. He complained of severe headache, was restless during the night, an exophthalmos appeared, he was incontinent and during his attack bit his tongue. His left arm went into a definite convulsive movement. This lasted ten minutes and he had a second attack later in the night. Was taken to the hospital and his mental condition cleared up during transportation to the hospital. Two hours later he became somnolent, vomited and, although he seemed clear mentally, he occasionally could not find the word for which he sought. Examination September 26: General examination negative. Skin negative. The scleræ show a slight subicteric tinge. The pupils were slightly unequal, possibly somewhat irregular. The right pupil appeared to react somewhat slowly to light. Abdominal reflexes absent; two hours later, present. The left one was, however, the weaker. Sensation normal excepting in the left mammillary zone where sensation for sharp and dull was decreased. Definite rigidity present in

both arms, left more than right. Definite rigidity in the left leg. There was an area of decreased sensation to sharp and dull over the left tibia and dorsum of the foot. Lumbar puncture revealed a high cell count. There was a gold sol reaction in the first zone. Complete recovery by October 3. November 1 he again had fever, was unconscious and was taken to a hospital in Baden Baden, where a presumptive diagnosis of brain tumor was made, but because of the lack of a definite localization, no operation was undertaken. He was in coma for three days and died November 4, 1924.

No. 9, Gagel,[37] 1927. A boy eight years of age. After an attack of measles he developed a decrease of his mental faculties, was uninhibited and had a spastic paretic gait. The condition grew gradually but steadily worse. His intelligence decreased so that calculation, writing and reading became impossible. About three-quarters of a year after the onset there were high grade spastic phenomena in all four extremities. In about one year the patient stopped speaking. He did not react to external stimuli but only waved his arms here and there in an athetotic fashion. He then developed great difficulty in swallowing and died one and a quarter years after the onset, with pneumonia. His previous history had been essentially negative.

No. 10. Foix and Marie,[7] 1927. Patient, aged 63, had a spasmodic clubfoot since infancy. This condition appeared with convulsions at the age of a few months, after which the deformation of the right foot appeared. The foot at the time of examination was in the varus equinus position. There was a limitation of movement in dorsiflexion of the foot, although strength in the anterior leg group of muscles appeared normal. There was a considerable muscular atrophy, more marked in the extensors of the legs. Knee jerks active; the Achilles reflex could not be elicited, probably because of the contracture. Babinski sign positive. Anatomically there was found an era of centro-lobar localized sclerosis at the upper part of the left cerebrum. This sclerosis ramified throughout the considerable part of the left cerebral hemisphere's white matter. Microscopically it consisted of adult neuroglia tissue and Foix and Marie are inclined to interpret the condition as an encephalitis periaxialis diffuse which has come to a standstill.

No. 11. Liebers,[8] 1928, has summarized the clinical history of a patient, previously described by Merzbacher under the name of aplasia axialis extracorticalis congenita. The case was that of a girl who came from a remarkable family in that all told it included fourteen known cases of the disease. In the case of the patient in question the disease began in the third or fourth month of life with shaking of the head and eyes. Then there appeared gradually increasing spastic contractures of both the upper and lower extremities with positive Babinski and a fixed expression to the face, nystagmus, scanning speech, tremor and choreo-athetoid movements. Because of the increasing contractures and an osteoporotic involvement of the skeleton with deformities of the thighs, the patient finally became permanently bedridden. There were no sensory disturbances. The intelligence was only slightly involved. Death came from an intercurrent pulmonary involvement. While Liebers keeps this case separate from the other cases of diffuse cerebral sclerosis and designates it as the Pelizaeus-Merzbacher disease, it seems to me more likely that the condition is simply one in which the heredo-familial factor stands out extremely prominently. The pathology conforms fairly well to that described for the other cases of Schilder's disease.

No. 12. Bielschowsky and Henneberg,[10] 1928. Case 1. The birth and early development of the patient, a boy, were normal. Without recognizable cause he became ill at the end of his eighth year of life, with decreased hearing and spasms in the legs. The course of the disease was rapidly progressive. After two months a high grade decrease of vision already existed due to optic atrophy, and there were also severe spasms in the lower extremities. The reflexes were bilaterally spastic, more marked on the right side than on the left. After two months more there was blindness and severe loss of hearing. It seemed, however, that there was greater difficulty in the understanding of words than actual decrease of auditory acuity. Then rapidly appearing dementia occurred and the patient stopped playing, he cried incessantly, was incontinent, and ceased speaking. He also deteriorated markedly from a physical standpoint. Eleven months after the onset he was in a terminal state and there were severe spasms of the arms and legs, fixed pupils, optic atrophy, loss of the corneal reflexes. Spasms were more pronounced during spells of crying. Blood and the spinal fluid were normal. Death occurred in a cachectic state after a period of illness of not quite one year.

No. 13. Bielschowsky and Henneberg,[10] 1928. Case 2. The patient was the brother of the former patient. Normal birth. As a small child had attacks of laryngospasm and also temper tantrums, "Wegbleiben." Later there were pavor nocturnus and somnambulism. The disease began at ten and one-quarter years of age with decrease of hearing. Also in this case the auditory aphasia seemed considerably more marked than the decrease of hearing itself. Nine months after the onset there was disturbance of vision evidently due to optic atrophy. After one year spasms appeared in the lower extremities. At that time the mental condition was still good. Trepanation and inunction treatment had little effect on the course of the disease. One and a half years after the onset there appeared a series of epileptic attacks followed by a condition of unrest and confusion. During the months just previous to death the following symptom-complex appeared: blindness with advanced optic atrophy, complete deafness, extensive feeblemindedness, spastic paresis in the extremities, athetoid movements of the arms, increase of reflexes (though there was at the same time absence of the left knee jerk and decrease of the lower abdominal reflexes), periodic attacks of crying and laughing. Death occurred at the age of twelve and three-fourths years in a cachectic state.

No. 14. Sheldon, Doyle, and Kernohan,[38] 1929. Case 1. A boy aged 7, was admitted to the Mayo Clinic June 6, 1926. Ten weeks previously the parents had observed that the child was growing deaf and becom-

ing somewhat apathetic. Six weeks previously it had been noted that his eyes no longer converged and that he stumbled and seemed clumsy. Three days before admission he fell to the floor. He rose only to fall again in a similar manner. The legs began to twitch and the arms and face were successively involved, while the eyes turned upward and to the left. Convulsions were controlled by ether. On regaining consciousness after the anesthetic he was blind and complained of frontal headaches which persisted for twelve hours. The following day he had another convulsion and then vomited about twelve times during the next twelve or fifteen hours. The vision and general condition then improved rapidly. On examination he was found to be partially deaf, but he also failed to understand much of what was said to him when he could hear. He could read moderately large print. Perimetric examination could not be performed. Fundus negative. Neurologic examination showed slight vertical nystagmus when the patient looked to the left. Convergence absent. The pupils were dilated and reacted to light and accommodation. There was suggestion of partial paralysis of the lower portion of the right half of the face with associated movements of the fingers and toes on the left side. The right patellar and Achilles reflexes were somewhat exaggerated and Babinski's sign was present bilaterally. The abdominal reflexes were not obtained. Vibration sense was lost over both malleoli. There was incoördination on the finger-to-finger and heel-to-knee test, particularly on the left side. The gait was somewhat ataxic and slightly spastic. Spinal fluid negative except for a curve in the colloidal benzoin test in the middle zone. *X*-ray studies, urinalysis, blood counts, Wassermann reaction and urea content of the blood were all negative. The patient had a suppurative otitis media on the right side which cleared up under treatment. Condition was unchanged until July 12, when he vomited several times on arising, complained of severe headache and expressed fear of dying. He then had a slight general convulsion, was stupid and confused afterward and occasionally cried out as in pain. Several attacks followed in the course of the next few hours. Was taken to a hospital, where he appeared dull and exhausted though he tossed about restlessly. He soon became noisy and violently resistive, thinking he was being poisoned. Consciousness was partly regained the following morning. He was blind at that time. During the day he became more rational and the vision improved. July 16 preservation of speech was noted. He was more emotional. He was totally deaf and continued having convulsions. Ventriculography was performed July 30 and it was noted that the subcortical cerebral substance was resistant to the passage of the cannula. Grew steadily worse; by September 15 he was unable to walk; control of the arms and legs was gradually lost and about October 10 he became unable to speak. Early in November he had difficulty in chewing and in moving the tongue. Subsequently dysphagia developed. He died November 26, 1926.

No. 15. Sheldon, Doyle and Kernohan. Case 2. "A woman aged 27 was admitted to the Mayo Clinic June 29, 1927, complaining of blindness. She had always been well and strong. Six and a half weeks previously she had been delivered of her first child by forceps. Two days later, a foul vaginal discharge was noted, and fever and malaise set in and persisted for three weeks. Four weeks before admission the patient had noted slight blurring of vision which progressed to complete blindness in the course of a week. Subsequently her general condition improved considerably."

Examination showed complete blindness, dilated pupils, slight optic neuritis, partial paralysis of the lower part of the left half of the face. The laboratory tests were negative. By July 2, the patient became slightly stuporous and complained of nausea. She had a generalized tremor, the facial paralysis was more marked, the tendon reflexes were increased on the left side and she was involuntary. By July 5 she had a complete left-sided hemiplegia and was in coma. The spinal fluid pressure was 300 mm. of water. Cerebral abscess was suspected. She was operated upon July 5 and died July 9.

The gross pathology of this case does not conform to the ordinary picture of "Schilder's Disease" and certain features of the microscopic pathology are atypical.

THE CLINICAL AND PATHOLOGIC PICTURE

Schilder has summarized his investigation of 1912 as follows:

"1. First, there is a diffuse disease of the white matter of the cerebral hemispheres, occurring in childhood, which one is justified in considering as a separate disease on the basis of a characteristic macroscopic and microscopic pathology. I propose the name encephalitis periaxialis diffusa for this disease."

"2. The clinical picture of the disease is a very inconstant one. It occurs now under the picture of a brain tumor, and again under the picture of multiple sclerosis, and further under the picture of diffuse sclerosis of the type Heubner. One will naturally suspect the existence of the disease if the symptoms indicate an extensive lesion of both hemispheres. Rapidly developing cases of the symptomatology of multiple sclerosis at an early age of the patient will likewise suggest the possibility of this disease. The disease is fatal and the course has only a slight tendency to remission."

"3. Encephalitis periaxialis diffusa stands closely in relationship pathogenetically to multiple sclerosis, and especially closely to the acute forms of multiple sclerosis. Also for this disease there must be assumed a toxic effect on the brain. It has no relationship whatsoever to pseudosclerosis, tuberous sclerosis or to a certain number of the cases of so-called diffuse sclerosis."

"4. The etiology is still unexplained. Probably endogenous factors favor the development of the disease. The morphological findings do not support the assumption of an abnormal embryologic development of the brain."

Globus and Strauss[6] described the clinical group as follows:

"Thus an abrupt onset, a rapidly progressive course, characterized by mental deterioration, rapidly advancing spastic paralysis, epileptiform attacks,

features of decerebrate rigidity, ocular palsies and other ocular manifestations such as nystagmus, optic atrophy, and blindness without optic atrophy followed by a rapid decline with fatal issue (within two years) are fairly typical."

Collier and Greenfield[4] have outlined the clinical aspects of the condition as follows:

"A malady usually occurring in children and young subjects with no tangible causal factors or antecedent experience. The onset is a few days, the course progressive and some remission to a fatal issue, the duration from a few months to three years. The chief early sign is cerebral blindness which becomes complete, to which is added mental reduction and increasing spastic paralysis. Unsteadiness from parietal involvement and deafness from temporal involvement may be conspicuous. The amentia increases and passes into coma, which terminates the illness. The condition, usually bilateral, may commence on one side or may be confined to one side."

As Bouman's[3] observations and literature citations show, the foregoing statements of the clinical syndrome are inadequate in that so many other symptoms may appear that one is left with no very satisfying concept of the clinical picture of the disease. For instance headache, vomiting, incontinence and fatigue were found in the majority of the cases that Bouman reviewed. Epileptiform attacks were common. A few of the cases showed choked disk. Tonic and clonic convulsions occurred in several of the cases. Pupillary anomalies occurred. There was Argyle-Robertson sign in one. Strabismus, diplopia, disturbances of the conjugate movements were common. Transitory paralyses were noted. The tongue was commonly partially paralyzed. Intention tremor occurred. Then, too, while most of the patients are young, the condition is by no means limited to infancy or childhood.

Cases such as that reported by Bielschowsky[2] and reviewed in the present paper as No. 2 in the series reported from the literature, do not fit into the clinical picture at all as it is usually conceived.

It is evident in reviewing the literature of the condition that a clinical diagnosis of diffuse cerebral sclerosis can be made only in a certain percentage of the cases. The condition will frequently be confused with brain tumor, with multiple sclerosis and possibly with syphilis and arteriosclerosis.

Pathologically the findings in most of the reported cases conform reasonably well with those in the present material. However, the pathology of the condition as described by various writers is not always uniform. Sheldon, Doyle, and Kernohan's[18] case 2, Foix and Marie's[7] case and others show this lack of conformity. Of course, no two cases are absolutely identical, but in general, subcortical diffuse demyelinization with astrocytic glial overgrowth in the region, secondary degeneration of the pyramidal tracts and more or less preservation of the subcortical arcuate fibers are the principal pathologic characteristics of the condition.

NOSOLOGICAL CONSIDERATIONS

Several attempts have been made by various writers to properly classify the various conditions grouped together under the term diffuse sclerosis. Neubürger has proposed the following subgroups under diffuse cerebral sclerosis: First, a blastomatous or neoplastic; second, an exogenous inflammatory; and third, an endogeneous degenerative. As Bielschowsky and Henneberg have stated, however, the blastomatous group is a very weak one nosologically. While it is conceivable that a glioma might infiltrate a very large portion of both hemispheres of the brain, it is unlikely that such a tumor would limit itself to the white matter alone, producing degeneration of the myelin sheaths with relative preservation of the nerve fiber processes themselves, however, and then evidence itself as a sclerotic process, as scar formation, instead of showing the many types of young glial cells which one usually sees in a glioma. In other words those cases of so-called diffuse sclerosis which have been considered neoplastic are to be looked upon with a good deal of suspicion as being probably not neoplastic.

Schilder's term encephalitis periaxialis diffusa implies an inflammatory basis for the condition. His original case is in itself not at all conclusive either in the clinical course or in the pathological findings of an inflammatory condition. Probably the most outstanding case in the literature which could be classified as inflammatory diffuse cerebral sclerosis is the one reviewed in the present paper as reported by Bielschowsky. But even here, though the clinical findings are extremely suggestive, the pathological picture is by no means conclusive of an inflammatory process. Of the so-called inflammatory cases, while admitting the possibility of such a group, there is a dearth of good illustrative material and we are totally in the dark regarding any satisfactory designation

of the etiologic factors which may underlie the inflammatory production of the diffuse sclerosis in any one case.

To state that a condition is endogenous and degenerative is applying a name to it which does not carry much force unless we can indicate the factors, endogenous or degenerative, which produce the condition. The only obvious endogenous factor which has been satisfactorily pointed out in the literature of this condition is the hereditary factor. The case presented in the present article is probably a member of this group. Bielschowsky and Henneberg have reported hereditary cases. Ferraro, Krabbe and others have also given us examples belonging to this group.

Though one may have difficulties in a given case in the clinical differentiation of diffuse sclerosis from multiple sclerosis, with the rare cortical sclerosis such as has been reported by Rusk and Nixon,[19] with tumors and probably with certain other conditions, the clinical and pathologic picture as a whole is sufficiently definite to permit its separation from other sclerosing entities. In the light of what has been stated in the foregoing it seems to me that a simple terminology is still usable. The terminology should be on a descriptive basis because of our lack of knowledge of the etiologic factors except heredity.

A simple classification follows.

In order to designate the group of conditions falling under the general heading one might use the term *Diffuse Subcortical Sclerosis.* "Subcortical" is more accurately descriptive than "cerebral."

Under *diffuse subcortical sclerosis* then one could place three general types: 1. *Inflammatory type.* 2. *Hereditary* or *familial type.* 3. *Primary* or *essential* or *non-hereditary* or *sporadic type.*

1. The *inflammatory type* would include such rare cases as Bielschowsky's.[2] In those cases where the inflammation is due to a well-known etiologic inflammatory entity, the diagnosis of diffuse subcortical sclerosis should not be used, but the diagnosis should be that of the primary inflammatory condition.

2. The *hereditary* or *familial type* would include such cases as that reported in this paper, Bielschowsky and Henneberg's, Scholz's, Krabbe's cases and those of the so-called Pelizaeus-Merzbacher disease. Bielschowsky and Henneberg suggest the subdivision of this group into an *acute infantile* (Krabbe), a *subacute juvenile* (Scholz), and a *chronic type* (Pelizaeus-Merzbacher). In the light of the small number of cases and the lack of good definition between these forms it seems to me unnecessarily complicated to make these distinctions.

3. The *primary* or *essential* or *non-hereditary type* would include those cases of diffuse cerebral sclerosis identical with those of the *hereditary* or *familial type* but lacking the hereditary factor. The occurrence of sporadic cases of hereditary diseases is a common observation in any larger medical clinic. No doubt, therefore, many cases that might naturally be placed in this category really belong with the *hereditary* or *familial* type. Possibly, if our knowledge of family histories were complete, we would be in a position to do away with the *primary* type altogether.

SUMMARY

1. A case of diffuse subcortical sclerosis of the familial type is presented clinically and pathologically.

2. The case reports from the recent literature of this and allied conditions are summarized.

3. The confusion of classification and terminology is pointed out.

4. A simple classification is suggested as follows:

(a) The term *diffuse subcortical sclerosis* is a satisfactory designation for the general group. The subgroups are:

I. Inflammatory type.
II. Familial—hereditary type.
III. Primary or sporadic type.

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LOCALIZED ACTINOMYCOSIS*

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IN 1875 Cohn described a filamentous organism obtained from a lacrimal duct abscess, which he called a streptothrix. Bollinger in 1877, in collaboration with Hartz, the botanist, described a new fungous disease of cattle. Because of the ray-like arrangement of mycelial threads of the fungus, they called the condition actinomycosis.

Israel in 1878 reported from the Jewish Hospital in Berlin three cases of a chronic, localized, pyogenic disease in human beings. He gave an excellent description of the pathologic process and what he took to be the causative agent, a mycotic fungus with micrococcus-like bodies. These micrococci, he said, were either forms of the fungous growth or organisms which grew symbiotically with the fungus. Cohn was permitted to study Israel's organism and he identified it as the same as that described by him three years before.

Eppinger in 1890 studying the cultural characteristics of the ray-fungus, attempted to find evidence of the production of a soluble endotoxin, but with negative results. Harbitz and Grondahl, in 1911, studied eighty-seven cases of actinomycosis giving special attention to the source and mode of infection. They found many types of actinomyces occurring in nature, but none resembling those recovered from the lesions in man, a specific anaërobic actinomycete (actinomyces hominus). Wright, Shiata and others came to the same conclusions.

The occurrence of five cases of localized actinomycosis in my own practice in two years suggested this presentation. In most of my cases the patient had to do with the handling of cattle, and two gave definite histories of the entrance of a piece of grass or barb of barley in the exact location where the lesion ultimately developed. It is quite possible that the infection is transferred from one infected animal to another through the contamination of grass or hay, the entrance of such a foreign body making an ideal anaërobic inoculation.

Actinomyces are found in nature and can easily be cultured from plants and grasses, or their spores recovered from the air. In every instance these organisms are found to be of the aërobic type, producing in their growth, aërial hyphæ, whereas the pathogenic type is constantly anaërobic and grows only beneath the surface of the medium. The pathogenic organism has been found only in its animal hosts, but the presence of bits of grass or hay in the lesions has been considered by many as conclusive evidence that these were the carriers of the infection.

The lesion of actinomycosis is a very constant one, the focus of infection always essentially a granuloma. Depending on the age and location of the granulomata, they vary in size from that of a pea to large irregular masses often connected with one another by fistulous tracts lined with the same tissue. The lesions contain very little fluid pus and almost no necrotic tissue. The amount of tumefaction about the lesions is dependent on their location. If situated in the neighborhood of fascial planes there will be an enormous amount of fibrous tissue converting the entire region into one homogeneous mass. On making a section through such an inflammatory area it will be found to be made up of the various types of tissue, such as muscle, fibrous tissue, and, when in that location, salivary gland. The infiltration with new-formed fibrous tissue is of such uniform hardness that the normal cleavage planes are entirely obliterated. Somewhere within this mass will be found one or more granulomatous masses made up of soft, red, pulpy granulation tissue, and, if there is no secondary infection, only a slight amount of pus. If one searches diligently an occasional sulphur granule will be found.

Obviously the symptoms vary according to the location of the lesion. Patients ordinarily suffer very little from the presence of such an inflammatory process. In lesions about the face or neck there may be neuralgic pain from involvement of the sensory nerves, or stiffness of the neck or trismus if the muscles of mastication are involved. Variation from the normal blood-

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picture depends entirely on the presence or absence of secondary infection.

The most frequent type of infection is the cervico-facial form. In the typical advanced case of this type of infection the inflammatory mass extends from the parotid region into the submaxillary triangle. The swelling of the neck often extends beyond the line of the jaw, the skin being shiny, purplish in color, and the entire involved tissue of wooden hardness. There may be superficial, fluctuating areas or depressed fistulous openings, and often scars from previous incisions. In lesions as extensive as this there is regularly trimus from involvement of the muscles of mastication.

In regard to the diagnosis of actinomycosis, it may be said that any indolent, chronic, indurative, inflammatory process should be under suspicion, but the only positive diagnostic finding is the discovery of the ray-fungus in the sulphur granule. These sulphur granules are not as common as one might expect, and we have seen cases that were treated as being actinomycosis, with a moral certainty that the diagnosis was correct, in which sulphur bodies were never found. In one instance, the first time the patient was seen a sulphur granule was expressed from the fistulous opening, and although the entire mass was radically excised and submitted to careful examination, additional ones were not discovered. Unfortunately, although these organisms can be grown *in vitro*, it is almost impossible to grow them from the tissue or any other material than the sulphur granule itself. Therefore, cultures are of no value, since, if the granule is found, the diagnosis can be made immediately by microscopic examination of the unstained material. To make the microscopic diagnosis one must be able to demonstrate the ray-like arrangement of the club-shaped bodies, for other large organisms, notably Vincent's, tend to form yellowish clumps. The long fusiform bacteria can easily be confused with the mycelial threads of the ray-fungus, especially if one is prejudiced.

The treatment of actinomycosis should be a definite and very radical procedure, and the clinical and pathological pictures are quite constant. Even if the fungus cannot be found, I believe that in such an instance it is justifiable to make a clinical, unsubstantiated diagnosis and operate accordingly.

In discussing the treatment of this condition I have in mind the cervico-facial, cellulitis type of lesion, chiefly because this form lends itself to surgical treatment. The actinomycete is strictly anaërobic and not vigorously pathogenic, and if a single granuloma is given a thorough communication with the air, I believe it will be destroyed by this procedure alone. It is next to impossible to locate these areas in the large masses of indurated tissue, and, unlike collections of pus that lie in fascial spaces with normal cleavage planes leading to them, the actinomycotic granuloma is in a solid mass. The only certain method of eradicating it is the radical excision of the entire involved area in one or more stages. I have always made this excision with a knife, but I believe the next one I have to do I will try the high frequency knife to obviate the bleeding. The area to be excised should be marked out in the neck and the incision so planned to avoid, if possible, the branches of the facial nerve. The wound should be funnel-shaped to an exaggerated degree. The bleeding can be best controlled by packing, as one goes along, with absorbent cotton. If the indurated tissue extends behind the mandible this too should be excised. It is better to get beyond the tumor in one part and leave the remainder for later stages, than to do the entire operation in a perfunctory manner. After the excision, if the bleeding is severe, packing with dry cotton for a short time will usually control it. After the excision has been made I have packed the wound with equal parts of Lugol's solution and glycerin. This packing should be changed every other day. If the wound has been properly made and there are no undercuts nor crypts, this packing will come out quite easily and without pain, but the process of inserting the iodin packing is frightfully painful. The pain can be avoided by first packing the wound lightly with cotton soaked in 5 per cent cocain solution. This should be left in for five to ten minutes. The wound can then be packed with iodin gauze. (I use cocain this way very often in doing painful dressings and exploring fresh traumatic wounds and have never seen any constitutional effects from it, although I would hesitate to use the same solution on mucous surfaces.)

If in a week or ten days after such an operation, a portion of the wound has failed to show a sufficient degree of resolution, radical excision should be performed.

I am not sure that potassium iodid given internally has any value. We have given it in increasing doses to all our patients, but favorable results followed only in conjunction with surgical procedures.

We have tried *x*-ray and radium, but I will admit that the cases in which we tried them were not fair tests of the treatment.

REPORT OF CASES

Case 1.—Mr. A. K., a rancher, thirty-two years of age, entered the Miller Hospital on January 26, 1929, with complaint of a discharging sinus in the left side of the neck and jaw. The present illness had begun five months previous to hospital entrance, when the patient got a foxtail grass under his tongue. About ten days later a small lump appeared in the left side of his jaw. At the end of three weeks it was lanced, only a very small amount of pus being obtained. The wound was packed with iodoform gauze until it closed. It again increased in size and six weeks later was reopened. It healed the second time. The induration increased and shortly before admission to the hospital it broke down and discharged of its own accord.

Past history was negative and the family history was not pertinent to the patient's condition. Social history brought out the fact that he had looked after stock all his life, but there was no history of actinomycosis in his cattle in recent years.

Physical examination was negative except for chronically infected and hypertrophied tonsils and the essential pathology, which was a mass on the left side of his neck extending from the parotid region to the midline and filling the anterior cervical triangle. There was an opening in the most prominent part of the tumor, which exuded considerable pus, and scars from previous operations were evident.

The blood and urine were normal.

The day after admission a complete excision of the mass in the anterior cervical triangle was made under ether anesthesia. The wound was packed with gauze soaked in 50 per cent Lugol's solution and glycerin. This was changed frequently, the wound first being cocainized before insertion of the iodin gauze.

Twenty days after operation the wound had almost healed, and a tonsillectomy was performed under local anesthesia, without complications.

Patient was discharged thirty days after admission and has been subsequently reported as well.

Case 2.—Mr. R. R., railroad executive, forty-two years of age.

The patient rode horseback but had nothing to do with the care of animals nor with cattle. Three months before admission, while driving in the country, he stopped at a farm house and while walking through a lane picked an ear of grain and held it in his mouth. One of the barbs from the grain stuck in the posterior part of his tongue. He made several futile attempts to remove it and was still conscious of its presence the next morning, when he consulted a nose and throat specialist, who was unable to remove the foreign body. When I saw him, approximately three months later, he had a tumor of the tongue just anterior to the lingual tonsil. Incision was made into it and with a curette some soft, red, friable tissue was removed. There was one small mass of pus which contained a typical sulphur granule in which the ray-fungus was identified. The incision over the mass was enlarged so that the wound was as large as the widest part of the granuloma. It was packed with gauze soaked in Lugol's solution and glycerin every day until it entirely healed. At the end of two weeks it had entirely healed and there has been no recurrence.

Case 3.—Miss F. K., a clerk, twenty-seven years of age, was admitted to the Miller Hospital on June 27, 1922, with a complaint of soreness and stiffness of the tongue and difficulty with speech. Present illness began five months previous to hospital entrance, when she noted that her tongue felt stiff and sore and ached a great deal. These symptoms would be aggravated by attacks of tonsillitis. Shortly before admission a tonsillectomy was done, but with only slight improvement. One week before entering the hospital a small, dark red spot resembling a blood blister appeared near the midline of the dorsum of the tongue about one inch from the tip. This spot persisted. She also complained of headache, nausea and frequent colds.

The family and social histories were essentially negative. Past history was negative except for frequent attacks of quinsy and sore throats since she was twelve years old.

Physical examination showed on the dorsum of the tongue a small fistulous opening directly in the midline, about which there was some induration resembling a cylindrical mass extending from the dorsum of the tongue to the region of the hyoid bone.

Under local anesthesia an elliptical incision was made on the dorsum of the tongue around the fistulous opening and the cavity exposed would admit a finger. This was filled with dark red granulations. It extended as far as the hyoid bone, apparently in the substance of the hyoglossus muscles. The cavity was thoroughly curetted and packed with iodin gauze.

The microscopic examination showed the curettings to consist of fibroblasts, lymphocytes and pus cells. There was one small round body surrounded by pus cells consisting of a mass of coiled, blue-stained filaments surrounded by a corona of radiating, red-stained clubs typical of the ray-fungus.

The patient was discharged as cured in four months and there has been no recurrence.

Case 4.—Mr. B. F. R., a live-stock commission merchant, thirty-five years of age, entered the hospital, complaining of a mass on the right side of the neck, stiffness in the neck and some pain.

Patient was a live-stock commission merchant at South Saint Paul and his work necessitated his being around stock and he was given to chewing hay. About one month previous to his admission a small mass appeared near the angle of the jaw. This gradually increased in size until it reached anteriorly almost to the midline. The patient's own diagnosis was that he had "lump jaw" and that it was caused by his habit of

chewing hay. His past history was negative.

Physical examination was negative except for a firm, hard, slightly tender mass on the right side of the neck extending from the angle of the jaw toward the midline. Near the center of this mass was a small point of fluctuation.

At operation the mass was opened and a granulomatous cavity about the size of a hickory nut was found. Material from this mass showed the typical sulphur granule. The entire indurated area was then excised and packed with iodin gauze. The excised tissue showed one or two other granulomatous areas.

The wound healed entirely and his recovery was uneventful.

Case No. 5—Mr. O. M., a farmer, sixty-five years of age, entered the hospital on September 22, 1928, with a diagnosis of advanced actinomycosis in the cervico-facial region.

About three months previous to admission there was induration in the left submaxillary region. One month previous to admission patient developed pain and some dysphagia. His lower jaw began to swell, was hard and lumpy and very painful. This region was opened several times during the month before admission, but the process continued to extend.

In taking the family history it was learned that he had a son who died of a peculiar pneumonia that was preceded by a chronic abscess in the region of his jaw.

Physical examination showed an old man in fair health except for a hard, ulcerating, tender mass involving the entire parotid region and entire anterior angle of the neck. When pressure was exerted in the neighborhood of one of the fistulous tracts some pus with one sulphur granule was expressed. This was the only one we ever found.

The patient was operated on the day after admission and a fairly radical excision of the entire mass was made. The wound was packed with iodin-glycerin gauze. Two weeks later there was still a great deal of induration at the base of the wound and toward the angle of the jaw. A second operation was done, the excision being carried to the mandible, which was laid bare on its inferior and mesial aspects and the tissues excised toward the floor of the mouth. It was again packed with iodin gauze and hot, wet dressings were applied. This treatment was continued for a period of about thirty days, when he was allowed to go home under the same treatment.

A report from his family doctor indicated a gradual improvement and six months later his wound was entirely healed. Another six months showed no recurrence.

CONCLUSION

Actinomycosis is not rare. The fact that it is an anaërobic infection suggests its treatment. The sulphur granules are not numerous and may not be found. If this is the case, and the clinical picture is typical, the condition should be treated as though it were actinomycosis. The treatment should be radical excision and, if necessary, multiple operations until resolution of the induration is certain. Local treatment of the wound with iodin is probably of value. Frequent dressings with repacking of the wound is necessary to provide an unfavorable anaërobic situation.

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DISCUSSION

DR. JAMES F. PERCY (Los Angeles, Calif.): I would like to say a word in reference to the treatment of this condition. Dr. Zimmermann spoke of the high frequency on cures. If he will use the cautery he will not only clear up the cases very quickly, but he will have no pain and no hemorrhage. The high frequency will leave a sore or a surface that does not heal well, and it has practically no effect on the fungus growth.

I have a rule that can be followed in these infections, just as I followed it in cancer, in using the cautery: All tissues normally movable fixed by the pathology make normally movable again with the heat. This rule applied in this situation will clear up these cases very quickly.

DR. W. M. MILLS (Topeka, Kans.): I would like to ask if the Doctor places any value on the sustoxin treatment, or the iodid treatment.

In connection with Dr. Percy's statement, I had one case of actinomycosis of the palate, which was treated extensively by radium, with no results whatever. So I used a cautery and got prompt results. The infection was entirely healed in three weeks.

DR. W. B. HOLDEN (Portland, Ore.): I was wondering if anyone had used thymol in the treatment of actinomycosis, as brought out by Dr. Myers, at the University of Oregon. He has published somewhere—I can't tell you where—some very remarkable results in the use of thymol internally and locally in the treatment of actinomycosis.

DR. A. L. BLESH (Oklahoma City, Okla.): I will say that my first experience with actinomycosis occurred when I was a boy on the farm, in the disease known as "lumpy jaw" in cattle. The cautery was extensively used in those cases by the farmers themselves and veterinaries, without curing the cases as they occurred in the animals.

My own experience in the treatment of actinomycosis has not been extremely satisfactory. I have treated most of my cases with iodin in some form or other.

One particular case I recall I had a great deal of trouble with. I sent the patient to Rochester, and they sent him back without operating. So I really am gratified to know that Dr. Zimmermann has been sucessful and I shall try to take some lesson home with me on that score. I observe, in his discussion of the treatment, that in some of the cases he has merely corrected the condition, and then packed it with Lugol's solution. I wonder if he reached all the germs or all the infection by his operation or whether in this instance he intended the solution to cure the condition. I would like to have him bring that out.

DR. F. E. CLOUGH (San Bernardino, Calif.): I would like to ask one question relative to the use of copper sulphate internally and externally: Is it extensively used?

DR. ROWLAND H. HARRIS (Battle Creek, Mich.): I want to report two cases of actinomycosis that I have had: One was the case of actinomycosis of the tongue, occurring at the tip of the tongue, a mass about the size of a hickory nut. I made a clean incision and suture of the tongue and cleared out the granuloma under the jaw. The man is still alive, after five years, with no signs of any recurrence. I did nothing but the excision.

The other case is a woman upon whom I operated about twelve years ago for the removal of her appendix. I did not get the diagnosis then of actinomycosis. Several years later I operated again and removed the right ovary. I removed a mass the size of a goose egg that was actinomycosis. Two or three years later she developed actinomycosis on the left side, probably from some left over from the right side, and an abscess formed in the groin. She was given large doses of potassium iodid. She is still alive and quite well.

DR. GATEWOOD (Chicago, Ill.): I left two cases of actinomycosis when I left Chicago, and I expect a third one when I get back. One of them was a gas case; one was an ileocecal type, either involving the subphrenic region or the abdominal wall.

A word about diagnosis. Dr. Zimmermann has brought out the fact that the clinical picture is characteristic. It is so characteristic that one can make a diagnosis in spite of the laboratory findings. The best laboratory method we have found is biopsy. When repeated microscopic sections and cultures fail, biopsy of some of the granulated tissue will show the fungus in the tissue.

In regard to the use of copper sulphate, Dr. Zimmermann will answer that from his experience, but I can say a few words about it from our experience. The injection of copper sulphate makes a great many people sick, and I have been very doubtful as to its effect. I think potassium iodid possibly does more good internally, in the way of local application. Whether it is of benefit or not depends upon the kind of glasses the man wears: If they are blue, it cures them; if they are plain, it doesn't.

There is one other thing in the way of treatment that has not been mentioned, which I cannot pass by, because Monterey has the credit of curing at least one case: A patient who had been operated upon a number of times went from 160 pounds down to 90 pounds, and was given up as a hopeless proposition, the lesions being too extensive for a radical operation. That patient came here to Monterey and, living out-of-doors with nothing on, returned to Wisconsin at the end of two years' time, having regained her normal weight, without any other treatment.

DR. ZIMMERMANN (closing): With regard to the general treatment: We have used potassium iodid in all of our cases, but have not had favorable results. We have one Chinese patient with pulmonary actinomycosis who has had it for a year, and he has been spitting up sulphur granules and a great deal of pus. He had everything, but I blame the potassium iodid. He apparently is slightly improved but he still has *x*-ray evidences of a rather extensive lesion. He is not spitting any more of the granules and his sputum is a little bit less. I am sure that he will die of actinomycosis, although apparently he is somewhat improved.

With regard to thymol: I haven't tried it, but the reports do not look very convincing. Copper sulphate was tried some years ago; my partner tried it in one or two cases and got no results. Also, the literature on that was not very convincing.

I feel about actinomycosis as I do about cancer—nothing can be done by surgery that is half as bad as what they will do themselves eventually.

A wide incision, leaving the wound widely open, dressing frequently, and keeping it exposed is, I think, the crux of the treatment.

In regard to the suggestion that the cautery might cure actinomycosis because it has been tried on animals: I had an interesting experience in trying to work out the bacteriology of this thing. I sent to the stockyards for actinomycotic jaws of cattle. I got several specimens that were condemned as being actinomycotic. None were. They were specimens of lymphadenitis in the region of the jaw. I think the farmers who are around cattle are inclined to call any lump about the jaw actinomycosis. Lymphadenitis does occur in cattle.

Of course, with regard to the dissection of lymph nodes—it is not a question of lymph node dissection, it is a question of a wide excision of a large iflammatory area. The actinomycosis does not extend to the lymph channels, but it grows in the tissues and makes these granulomatous areas.

CASE REPORTS

BILATERAL SPONTANEOUS PNEUMOTHORAX

REPORT OF CASE

JOHN G. LAMONT, M.D.
Nopeming, Minnesota

This condition has been described as a fatal complication in pulmonary tuberculosis by Meyer, Marshak, E. A. Gray, Bourne, Greshey, LeWald, Elte, Benedict and Donahue.

Emmerson and Boeler and also Benedict report the complication as a cause of death in asthma. Briggs cites a similar result in a metal worker in whom autopsy revealed gastric carcinoma without evident metastasis to lung tissue.

Recovery has been reported in only five cases, exclusive of the present instance. Hawes, Jr., Gershey and Hazen each cite recovery from the complication in pulmonary tuberculosis, Eberts in a bilateral pyopneumothorax, and Bedford and Joules a complete recovery after fourteen days, in a patient otherwise healthy. In all patients who have recovered, some form of pleural deflation was used.

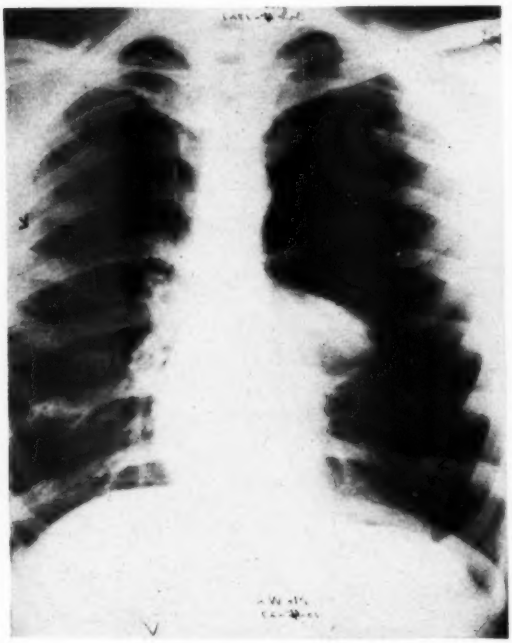

Fig. 1. October 4 (day following collapse). Lung margins right and left are indicated by arrows. Left upper lobe forms a dense shadow close to mediastinum.

Fig. 2. October 5. Second day. Skiagram following aspiration of 1,500 c.c. of air from left pleura. Left lung is partially expanded; heart and mediastinum drawn to left of former position; apparent increase in right pneumothorax.

CASE REPORT

On October 3, 1927, W. L., a young North Dakota farmer, was employed in pumping water with a double-cylinder hand-pump, into a water tank used to supply a steam threshing engine. Each tank filling involved a half hour of hurried labor. When the tank was almost filled, he was seized with pain in the left side of the chest, severe dyspnea and faintness. He reclined upon the top of the tank, and shortly, finding breathing less difficult, reached for the reins, and while still lying down, succeeded in driving his team and tank to the machines about a quarter mile.

He was carried into the farm house, and received medical attention from Dr. F. K. Kolb of Granville, North Dakota, who conveyed him next day by auto to the North Dakota Tuberculosis Sanatorium, a distance of seventy miles.

The first plate (Fig. 1), taken upon admission, indicated almost complete collapse of left lung, and a well defined air-space on the right side. The left upper lobe formed a dense mass close to the mediastinum, and moved freely with the cardiac impulse. There was moderate cyanosis, and rather profuse perspiration. Pulse 98, respiration 36, mouth temperature 98°.

Family History.—Father and mother are living, other

members of family healthy. There is no history of tuberculosis contact.

Personal History.—He had always been well, except for occasional gastric disturbance, and had always lived and worked with his father on the farm. He had had no infectious diseases of childhood.

Examination.—The patient is a white male, well nourished and weighing 145 pounds.

The entire left side of the chest presents a hypertympany with absence of respiratory sounds and a

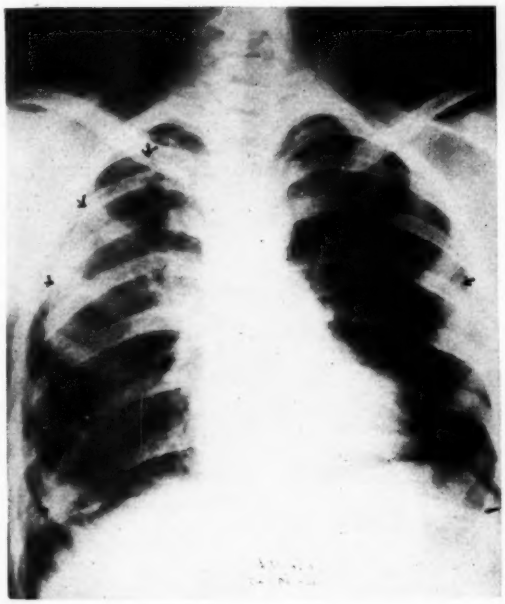

Fig. 3. October 10. Left lung is more fully expanded; heart in better position; right air-space smaller; atelectatic areas in right lung field.

Fig. 4. November 25. Left lung is expanded; right lung field and air-space more completely obscured.

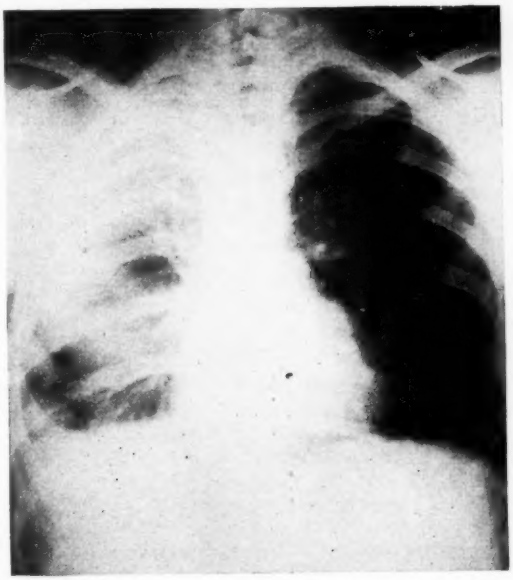

Fig. 5. December 29. Partial clearing of right lung field. There is complete expansion of left lung; small air-space in right costal angle.

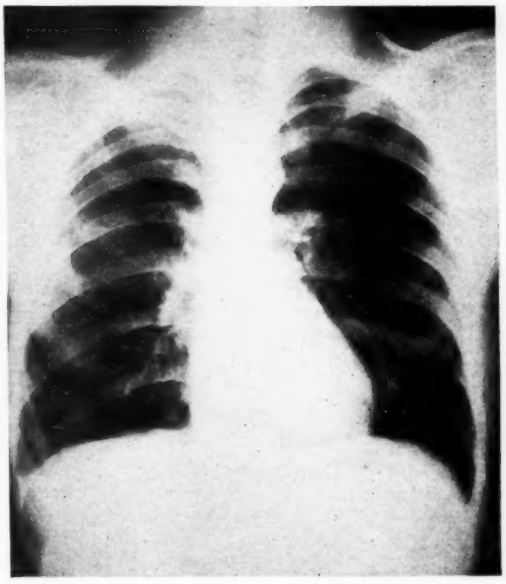

Fig. 6. September 21, 1930. Three years following attack. There is diffuse fibrosis in right upper lobe and left apex.

definite bulging of the chest wall. The respiratory murmur on right side is distant, except posteriorly. No râles are heard.

Course.—October 5, 1927, the left pleural space was aspirated in the eighth interspace, with an ordinary pneumothorax needle, and vacuum bottle. After 1,500 c.c. of air had been withdrawn, the operation was discontinued, because of a spasm of coughing. There was prompt relief from dyspnea, and Plate II was made within an hour, which indicated moderate expansion of the left lung, improved heart position, and slight increase in the right pneumothorax.

For several days, the patient was fairly comfortable, and no further deflation was attempted. The temperature ranged, however to 101.8 and pulse 96, and October 10th a skiagram (Fig. 3) showed clouding of the right lung field, with areas of atelectasis. The left lung maintained good expansion. There was no apparent increase in the pneumothorax on the right side.

Until October 22, the daily temperature ranged 100 to 102.8 with much restlessness, relieved by opium.

Severe dyspnea recurred suddenly on the morning of October 22nd. Aspiration of 1,500 c.c. at 3:30 p. m. from the left side gave much relief. The temperature fell during this day below normal, and, except for a slight evening rise, continued so, during later convalescence. On October 23, 900 c.c. and 1,000 c.c. were again removed. Gradual improvement continued during November and December. On November 25, the patient was able to visit the *x*-ray room. The *x*-ray indicated dense clouding of entire right lung field (Fig. 4). This showed moderate clearing on December 29 (Fig. 5).

Patient was allowed to leave the sanatorium on January 6, 1928, in good physical condition. The sputum, always scant, was repeatedly negative for tubercle bacilli. Von Pirquet skin reaction was positive. Following a report from Dr. Kolb in September, 1930, that the patient was doing ordinary farm work, at home, a request was made for a skiagram. The view shown in Figure 6 was taken September 21, 1930, and indicates only a moderate degree of diffuse fibrosis in the right lung, and left upper lobe. Patient states that he feels entirely well.

Comment.—Bilateral spontaneous pneumothorax may occur in apparently healthy individuals, following unusual exertion. It is commonly a complication of tuberculosis, or other conditions resulting in localized emphysema, with accompanying attenuation of the adjacent pleura. No parenchymal lesion due to tuberculosis was evident in the case above described. It is improbable that air passes from one pleural space to the other, as the unilateral form rarely becomes extrapleural under positive tension.

Deflation by use of the reversed pneumothorax apparatus is of value, in selected cases.

Acknowledgment is made of the use of the *x*-ray files and records of the North Dakota Tuberculosis Sanatorium at San Haven, N. D.

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SUBACUTE BACTERIAL ENDOCARDITIS INVOLVING THE TRICUSPID VALVE ONLY*

REPORT OF CASE

HARRY OERTING, M.D.
Saint Paul

The case I present this morning is that of an adult white male, aged 46, who was admitted to my service at the Ancker Hospital, St. Paul, on October 27, 1929, complaining of fever, chills, cough, vomiting and pain in the chest of three weeks duration. The onset, October 6, 1929, was extremely sudden and violent, with chills and a high fever. He was very ill for two or three days and since that time he felt only fairly well, and the cough and pain in the chest did not improve very much. Three days before admission he began to have nausea and vomiting.

The past history is negative except that he has always been subject to frequent colds and has had pneumonia several times.

The positive physical findings on admission were dental caries, marked pharyngeal injection, mild cervical adenopathy, dullness over both lower lobes of the lungs, the left more than the right. The breath sounds were harsh, with many râles over the right side posteriorly. There was bronchial breathing over the left lower lobe. There was a friction rub over both lower lobes posteriorly. The heart was moderately enlarged, rate 120, and the tone quality was rather poor, but otherwise there seemed to be no abnormality. No murmurs or bruits were heard. The abdomen and extremities were negative. The temperature was 100 F. and the blood pressure 115/90. The tentative diagnosis on admission was bronchopneumonia, pleurisy with effusion and a questionable empyema.

The laboratory data follow: Urine 1:020; albumin a faint trace with occasional hyaline and granular casts and a few blood cells. Stool negative. Wassermann positive. Blood chemistry: creatinine 2.8 mg.; urea N.

*Presented before the Minnesota Society of Internal Medicine, Duluth, Minn., May 26, 1930.

160.3 mg. Blood: Hgb. 59%, r.b.c. 3,190,000, w.b.c. 12,000. *X*-ray: Heart definitely enlarged, 6.5/11.3, comprising 58% of the chest diameter. Lungs: Infiltration at both bases which may be inflammatory but is probably due to congestion. There is a thickened interlobar pleura on the right between the middle and lower lobes. Possibly a resolving pneumonia. Sputum negative for Tbc. On October 31st thoracentesis was done and 40 c.c. of a clear straw colored fluid was removed which showed nothing microscopically.

The general condition remained about the same and daily observations failed to reveal any heart murmurs. On October 31st the patient became irrational, the temperature rose to 101, the white blood count was 17,800. The physical findings were not unlike those on admission. The spleen and liver were not palpable and no petechial spots were present. The condition remained about the same until November 4, 1929, when there was a sudden change. The breathing became very labored, the pulse very weak, the liver was definitely palpable, the spleen was not felt, the lungs showed a diffuse patchy tubular type of breathing over both lungs posteriorly. Numerous petechial spots appeared over the back, chest and extremities. The heart tones were rough and slurred but there was no murmur. At this time a diagnosis of subacute bacterial endocarditis was made with no definite idea of which valve was involved; bronchopneumonia; left pleural effusion; congestive heart failure.

On the afternoon of November 4 the patient died and the following are the essential autopsy findings:

Pericardium normal.

Heart.—Weight 400 grams. Mitral and pulmonary leaflets are normal in appearance. The aortic leaflets are sealed together at the commissure but there is no evidence of an acute vegetative lesion. The tricuspid leaflet shows a large thrombotic vegetation which measures about 3 × 1.5 cms. This is situated on the posterior tricuspid leaflet. It is soft and friable. Scattered over the balance of the valve there is a rather diffuse vegetative lesion which is flat and fairly firm. This does not resemble the small nodular type of lesion seen in pneumonic endocarditis. There is no evidence of luetic aortitis. The coronaries are normal. There is no gross evidence of excess fat or fibrosis. The myocardium is flabby and after cutting is found to be pale and swollen in appearance.

Pleural Cavities.—The left pleural cavity contains about 500 c.c. of clear straw colored fluid.

Lungs.—Consolidation along the posterior border of the right lung. Scattered throughout the lung are numerous nodules with a firm consistency and situated beneath the pleura. On cutting, these nodules are yellowish in color and well defined from the balance of the lung tissue. The left lung shows consolidation involving the lower portion of the upper lobe and the posterior portion of the lower lobe.

Spleen: The spleen weighs 94 grams. There are several well defined areas of infarction.

Kidneys.—There is no evidence of infarction.

Post-mortem Diagnosis. Subacute bacterial endocarditis (tricuspid). Multiple infarcts of the right lung and spleen. Bronchopneumonia, bilateral. Splenomegaly. Meckel's diverticulum. Pleural effusion (left).

L. Sogge, M.D.
President, Minnesota State Medical Association

EDITORIAL

MINNESOTA MEDICINE

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Vol. XIV January, 1931 No. 1

DOCTORS' BILLS

It is perhaps not strange that the medical profession in Minnesota is not greatly impressed with the cry that is heard so frequently about high doctors' bills. The fee schedule varies greatly and in Minnesota is very moderate compared to that in the large eastern cities.

Are doctors' fees so exorbitant? Compare them with those of an attorney in the same community. Or, compare the sum total with the sum spent for tobacco, cosmetics, radios, automobiles —all of them less important commodities.

The main difference, however, in medical expense compared with other expense, is that no provision is made in the family budget for medical expense, and the total cost to the nation is not evenly distributed. Inquiry would show that there are few individual accounts that run over $300 a year. A fee of this amount, however, would represent only a portion of the total cost of any particular illness. It must be admitted that in an appreciable, though small, percentage of instances, the financial burden due to serious or protracted illness is a heavy burden.

An article by the Director of the Twentieth Century Fund* offers food for thought. The author praises the scientific attainment of the profession but deplores its lack of organization in rendering medical service. He sees great opportunities for economizing in group practice. We doubt whether the existing medical groups can show any marked reduction in medical costs to their patients. Medical fees in any locality are much the same whether services are rendered by physicians as individuals or in groups. The author is keenly interested in the insurance plan. We have been and are. The author's recommendation is a combination of group practice and insurance by so-called guilds, thus combining the advantages of group practice and medical insurance in one unit. This guild would provide complete medical services to the families of members, including periodic examination for the detection of early disease, hospital and other medical services in case of sickness. It sounds well.

Various objections come to one's mind. There would be many opportunities for abuse of medical service furnished through insurance. The neurotic would be in his (her) glory. This individual would pester the life of the doctor more than at present. Every patient would demand day and night nurses and the most expensive room in the hospital, and minor illnesses might be prolonged inordinately. The individual would have to accept the services of the guild doctor and freedom of choice would not be possible. And, if freedom of choice as to medical attendant were permitted, the unscrupulous doctor could add to his fees. The main objection to the

*Clark Evans: A Cure for Doctors' Bills. The Atlantic Monthly, October, 1930.

insurance plan for meeting medical costs is that so far it hasn't worked very well. To be a financial success, the rates would have to be rather high—according to the author mentioned, some $200 per working man's family. The average working man would take the chance that sickness or accident would not come to his family or that his bills would not amount to that much in a year and would not take out the insurance.

Sufficient statistics are not as yet available to warrant insurance of this sort. The author cited refers to recent studies carried out by the Metropolitan Life Insurance Company which showed that families of working men spent, on an average, $140 a year for medical care. Two-thirds of the total amount was spent by one-fifth of these families.

Possibly the study being made by the Committee on the Cost of Medical Care, of which Dr. Ray Lyman Wilbur is chairman, will be able to present figures reliable enough to warrant the issuance of policies to cover, if not the total cost to a family, two-thirds or three-quarters of the cost. Such a policy might eliminate most of the objections to insurance as the answer to the high cost of medical care. Such a solution would certainly be welcomed by the medical profession.

THE USE OF TOXOID FOR IMMUNIZATION AGAINST DIPHTHERIA

Since the discovery by Behring in 1913 that active immunization against diphtheria could be produced in susceptible individuals by injecting small amounts of a carefully prepared mixture of toxin-antitoxin, much investigative effort and experimental research has been done to overcome the objectionable features of the procedure. The reactions to the early preparations of toxin-antitoxin were often so severe that it created in the minds of both physicians and laymen, if not a feeling of opposition, at least a feeling of reserve, regarding the routine use of the mixture for diphtheria prevention.

Park and Zingher, in New York City, by using considerably smaller amounts of toxin, were able to prepare a mixture of toxin-antitoxin which practically eliminated the severe reactions associated with the giving of the earlier preparations. In 1924, Zingher[1] reported the immunization of 91 per cent of a large group of susceptible New York school children, as shown by negative Schick tests six months after giving of three doses of 1 c.c. each of .1 L + toxin-antitoxin. No unfavorable reactions occurred incident to its use. Other clinicians have reported very much the same results.

Very largely as a result of the work of Park and Zingher, the use of 0.1 L + toxin-antitoxin for routine active immunization against diphtheria spread over the United States, with a marked reduction in the incidence of diphtheria in those areas where its use was prevalent. Coincident with the increased use of toxin-antitoxin for immunization appeared increasing numbers of reports of the occurrence of severe reactions to later therapeutic administration of horse serum. Due to the presence in the antitoxin component of toxin-antitoxin of small amounts of horse serum, many investigators believed that by the increased use of toxin-antitoxin an increasing number of children were being sensitized to horse serum. Other objections to toxin-antitoxin were the difficulties of preparations of the mixture, the difficulties of preservation of the material, the number of inoculations necessary to produce immunity, and the slowness of immunization, which required from four to six months to develop.

In 1923, while the very excellent work of Park and Zingher in this country was directed toward dilution of the toxin in toxin-antitoxin mixtures in order to overcome the undesirable reactions incident to its use, Ramon,[2] working in the Pasteur Institute and American Hospital at Paris, produced a modification of diphtheria toxin by the addition of a small amount (4 c.c.) of commercial formaldehyde solution to 1,000 c.c. of a standard diphtheria toxin preparation. When this mixture was allowed to stand for one month, at a temperature of 38 to 40 degrees C., it was found to be free of all the toxic properties of diphtheria toxin. At the same time it had the power to produce antitoxin, when injected into the body; the ability to retain these properties over a long period of time without special precautions for its preservation; and, further, the material contained no horse serum. This product was called anatoxin by Ramon. The same material, further modified and improved, is now more generally called toxoid.

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[1]Zingher, Abraham: Proc. Soc. Exper. Biol. and Med., 22:462, 1924-25.

[2]Ramon, G.: Paris Med., 20:480 (Dec. 6), 1924.

In 1928, Ramon and Helie[3] reported their experiences with toxoid to the physicians in America. Since their report appeared, a series of clinical investigations have been carried on by numerous workers, attempting to determine the relative merits of the antigenic properties of toxin-antitoxin and toxoid.

Recently Harrison,[4] under very carefully controlled clinical conditions, gave 355 Schick-positive children, ranging from one to sixteen years, three doses of 1 c.c. of toxin-antitoxin. Four months later, the Schick test was negative in 64 per cent of the cases. Different lots of stock preparations varied in degree of effectiveness from 35 to 73 per cent. For comparison, toxoid, routinely prepared, was given to another group of 476 Schick-positive children. Some of the group were given three doses of toxoid (.5 c.c., .5 c.c. and 1 c.c.) at seven day intervals; others were given two doses of 1 c.c. each at an interval of thirty-one days. Schick tests about four months later showed production of immunity in 95 per cent of the cases. Twenty-seven children over six years of age were found previous to the giving of toxoid to be sensitive to the diluted toxoid and these children were given toxin-antitoxin. No local or general reactions incident to the use of either toxin-antitoxin or toxoid were reported. The low average percentage of immunity production recorded by Harrison with toxin-antitoxin would probably have been higher had the Schick test been applied six instead of four months later.

From the accumulating evidence, reports indicate that toxoid offers the advantages of smaller dosage, that immunity can be established in a shorter length of time, in a higher percentage of cases, and there is no horse serum in the preparation to produce sensitization. A disadvantage to the use of toxoid is the reaction which may occur in children over six years of age. This may be determined by testing in a manner similar to the Schick test, previous to the giving of the toxoid. In case the individual is susceptible, toxin-antitoxin can be given without unfavorable reaction.

R. L. W.

[3]Ramon, G., and Helie, G. I.: Jour. Am. Med. Assn., 91:1028 (Oct. 6), 1928.

[4]Harrison, W. T.: United States Treasury Dept. Public Health Reports—U. S. Public Health Service, 45:1883.

PNEUMOCOCCIC SERUM

The serum treatment of pneumonia has had few enthusiastic supporters since it was first used before the war. The fact that the horse does not respond readily in the formation of antibodies to the various types of pneumococci has necessitated the administration of large doses of the unconcentrated serum and repeated intravenous injections. The serum reactions were so frequent and often so severe that the method has never become generally popular. The writer was not favorably impressed by a limited experience with this form of pneumonia treatment in an army hospital during the war.

Recently, however, reports have appeared in the literature, of series of type one pneumonia treated with a serum refined and concentrated according to Felton's method, showing a mortality reduction of ten per cent or more compared with control series. Serum reactions occurred in only ten to twenty per cent of the cases treated, which is much less than was experienced with the unrefined serum.

If one patient in every ten with pneumonia of type one can be saved by this serum, the method is worth while even though the serum treatment is expensive and costs $100 or more per patient. Even the most ardent advocate of the reduction of the costs of medical care would not think the price too great if it increased his chances of recovery ten per cent. The type one group of pneumonia is the most prevalent one, amounting to approximately a third of pneumococcic cases, and roughly, a third of these patients die.

The mortality from pneumonia and even that from the various types of pneumococcic pneumonia, is so variable in different localities, during various seasons, and in the different kinds of hospitals, that an evaluation of a therapeutic measure of this sort has been difficult.

The evidence now on hand, however, of the value of Felton's serum, merits the earnest attention of the profession.

HISTORY COMMITTEE

In these days of laboratory and machine-made diagnosis and treatment, it is difficult to realize how the generations preceding ours practiced medicine. Diagnosis and treatment were personal and an art. Reading the old works shows as

much keen clinical observation. That is often lacking nowadays. Who knows whether our first physician possessed a stethoscope or understood the art of percussion? The clinical thermometer was used in St. Paul for the first time about 1870 and we know who brought it here and have one of that era. Do you realize that the hypodermic syringe was not used in the United States till 1856 and that the use of the clinical thermometer and microscope is not mentioned in the Harvard Medical College catalogue until 1867-1868? A Minnesata physician in a case report published in 1856 stated that a microscopic examination of a tumor of the stomach showed cancer cells. Can you inform your Historical Committee anything more about the use of these instruments in the early days in Minnesota?

J. M. A.

OBITUARY

Dr. Frederick A. Dunsmoor

1853-1930

Dr. Frederick A. Dunsmoor, one of Minneapolis' outstanding surgeons for almost a half century, founder of the old Minnesota Hospital College and one of the organizers of the medical college at the University of Minnesota, died December 16, 1930, at his home in Hollywood, California, where he lived for the last five years.

Dr. Dunsmoor, who was 77 years old, was born in the Richfield district of Minneapolis. He attended high school in Minneapolis and the University of Minnesota. In early life he determined to go into the medical profession and at the age of 16 years entered the office of Drs. Goodrich and Kimball to study medicine. Subsequently he went to New York, where he was graduated from the Bellevue University and hospital.

Coming back to Minneapolis, he was in partnership for a time with Dr. H. H. Kimball. In 1877 he began to practise independently. About the same time, he accepted a position as professor of surgery in the St. Paul Medical College, where he taught two years. In 1879 he was county physician for Hennepin County.

Dr. Dunsmoor then accepted the chair of surgery in the medical department of Hamline University, which he held two years, resigning to devote himself to the organization of the Minnesota Hospital College.

He bought the old Winslow hotel on Main Street N. E., Minneapolis, and started the college in cooperation with several other physicians and citizens. Then, in 1884, he founded the first eye, ear and nose clinic in the Twin Cities.

He served as vice president and dean of the college besides being professor of surgery, surgeon to the dispensary and attending surgeon to the hospital. While he was in this position, the institution and the St. Paul Medical College were merged to form the medical college at the University of Minnesota, where he was appointed to the chair of clinical and operative surgery, which he held until 1913.

He had an active part in the development of Asbury Hospital and served as surgeon at different times at St. Mary's, St. Barnabas, Swedish and City hospitals. He was also surgeon at different times for more than a half dozen railroads.

Dr. Dunsmoor made it a custom to spend a short vacation each year in study in the great hospitals, colleges and scientific centers of this country and abroad. His articles on his specialties appeared in leading medical journals and he was recognized for his skill as a surgeon throughout the medical world.

Physicians who have worked with him not only praise his skill as a surgeon but also point out that he was always ready to give his service, whether or not the patient had the money to meet the expense.

He was a member of the Hennepin County Medical Society, the Minnesota State Medical Society, the American Medical Association, the Academy of Surgeons and various other medical and surgical organizations.

Dr. Dunsmoor was a charter member of the Minneapolis Club and the Commercial Club, now the Civic and Commerce Association. He was also a member of the Lafayette Club and the Hennepin Avenue Methodist Church, where he served in official capacities for many years.

In the early '80s Dr. Dunsmoor built a home on Tenth Street that was the show place of the city for many years. After the business district expanded to that locality, he moved to a home at Lake Minnetonka.

He was one of the pioneers in building summer homes in the northern part of the state. About 25 years ago he constructed an immense summer home on an island in Rainy lake, 15 miles east of International Falls. He named the island "Minisenot," which is the Indian word for "Home Island." The home is now the property of Dr. N. H. Scheldrup of Minneapolis, husband of a niece of Dr. Dunsmoor.

Dr. Dunsmoor was greatly interested in the beauty and health resources of that territory and at a hearing before the international joint commission at International Falls in 1925, was the first person to publicly suggest the development of an international park in that region which is now under consideration and which the American Legion and the Canadian Legion have suggested be made a memorial to men of the two countries who gave their lives in the war.

Dr. Dunsmoor moved from Minneapolis because of failing health, but he returned for frequent visits. He visited there last summer, at which time he spent almost two months at his old summer home at Rainy lake. He was then subject to heart attacks, of which his death was the result.

Surviving him are two daughters, Mrs. Homer P.

Clark, St. Paul, and Mrs. Frederick W. McCartney, Denver; a brother, Dr. John Dunsmoor, Los Angeles, and three nieces in Minneapolis, Mrs. A. W. Armatage, Mrs. Horace Lowry and Mrs. N. H. Scheldrup.

Dr. W. H. Hallowell
1853-1930

Dr. William Henry Hallowell, physician in Minneapolis for 47 years, died Sunday, December 14, 1930, at Rosedale Hospital following a heart attack.

Dr. Hallowell, who was 77 years old, was born in Philadelphia. He was a graduate of the United States Naval Academy at Annapolis, and an officer in the navy for two years. At that time he won medals in double sculls and as a member of Navy sculling crews.

He was later graduated from the medical department of the University of Pennsylvania, coming to Minneapolis in 1883.

He was a member of the Hennepin County Medical Society, and, before the medical college at Hamline University was consolidated with the University of Minnesota, was head of the obstetrics department there. He had a large charitable practice among the poor for many years.

Surviving Dr. Hallowell are a son, C. C. Hallowell, manual training instructor in the Minneapolis public schools; a daughter, Mrs. Helen Leach of Bremerton, Wash., and a brother, Dr. Charles E. Hallowell of Philadelphia.

Dr. Andrew E. Anderson
1859-1930

Dr. Andrew E. Anderson, one of the founders of Swedish Hospital, died December 11, 1930, at his home in Minneapolis.

Born in Sweden April 2, 1859, Dr. Anderson had been in the United States for 57 years and a resident of Minneapolis for 33 years. One of four doctors who started the Swedish Hospital, he had been on the staff ever since. He was a member of the Hennepin County, Minnesota State, and American Medical Associations. He was a graduate of the Colorado University school of medicine and started practice in Ironwood, Mich.

Surviving Dr. Anderson are his wife, a daughter, Bessie, Iron Mountain, Mich.; two brothers, Albert Malmgren, Marquette, Mich., and S. Malmgren, Ishpeming, Mich.

Dr. Dwight F. Brooks
1849-1930

Dr. Dwight F. Brooks, Saint Paul, though identified prominently with the lumber industry, retained his medical licensure in Minnesota. Born at Redfield, New York, June 10, 1849, he came to Minnesota when a boy of seven, when his father, also a physician, settled at Beaver, a small village in Winona County, Minnesota. Six years later the family moved to Minneiska, a thriving town in the same county. His father later entered the grain business and tried to dissuade his son from pursuing the study of medicine, but in vain.

Dwight Brooks, at the age of eighteen, matriculated at the University of Michigan, later attending the Long Island College hospital, where he received his degree in June, 1876. After practicing at Minneiska for a few years, his father's business enterprises claimed more and more of his time. Lumber came to be allied with the grain business and some fourteen branches of the business were established in Minnesota and North Dakota. When, in 1886, a yard was established in the Midway district in the Twin Cities, Dr. Brooks made his home in Union Park, Saint Paul.

In 1894 an alliance was made between his company and that of Scanlon, Gipson and Company, which later became the Brooks-Scanlon Lumber Company. Lumber activities extended to Oregon, Florida, the Bahama Islands, British Columbia as well as Minnesota.

In September, 1875, Dr. Brooks married Anna E. Keyes of Winona. A brother, Anson S. Brooks of Minneapolis, and three sons, Harry K. Brooks of Bend, Oregon, S. S. Brooks of British Columbia, and Edward Brooks of Minneapolis survive him.

MISCELLANEOUS

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

State of Minnesota *vs.* Olga Stenhoff

On December 2, 1930, the State Board of Medical Examiners revoked the massage license of Olga Stenhoff, formerly residing at 2021 Columbus Avenue, Minneapolis, Minn. The defendant was convicted on her plea to the charge of abortion on the third day of October 1930, in the District Court of Hennepin County. At that time the defendant was sentenced to serve not to exceed two years in the Women's Reformatory at Shakopee, Minnesota, where she is now imprisoned.

MEDICAL BOARD REVOKES LICENSE OF MINNEAPOLIS PHYSICIAN

On Tuesday, December 2nd, the Minnesota State Board of Medical Examiners revoked the license of Philip Mueller, physician and surgeon, with offices at 109 So. 9th St., Minneapolis. The Board found Mueller guilty of immoral, dishonorable and unprofessional conduct, the particular offense being the habitual indulgence by Dr. Mueller in the use of morphine sulphate.

This is not Mueller's first difficulty with the law. On June 6, 1913, the Supreme Court of Minnesota affirmed Dr. Mueller's conviction of manslaughter in the first degree. Dr. Mueller at the time served two years in the State Penitentiary at Stillwater. Dr. Mueller's conviction followed the death on May 17, 1912, of a married woman on whom an abortion had been performed.

A PAGE FORUM OF THE COMMITTEE ON PUBLIC HEALTH EDUCATION

Senator Jones and "Medical Socialism"

On December 8, the Chicago Journal of Comerce printed as its leading editorial an interesting comment on the Jones bill which its author wishes to bring to a vote in this session of the United States Senate. The editorial printed there in full was called "Medical Socialism." It is one of several editorials on medical matters that have appeared of late in the Journal.

"Senator Jones of Washington is seeking a vote on his bill for an annual appropriation of $1,000,000 to provide medical treatments for mothers and children," says this editorial. "Legislation of this kind has been enacted before but its life has been limited to a term of years. Always it has been argued that the assistance of the federal government in these enterprises was only a transient necessity. But Senator Jones would make it permanent. The federal government is permanently to join with the states in supporting doctors who provide medical treatment for expectant mothers, nursing mothers, mothers past the nursing stage, young babies, older babies and children as old as seven or eight years. Naturally, the $1,000,000 appropriation will increase and the medical activity of Uncle Sam will be expanded. The medical socialist bill of Senator Jones is part of the general socialist movement which is engulfing the United States."

The dark view of things taken by the *Chicago Journal* is not wholly shared by physicians themselves. They know better than any, perhaps, what is the real state of mind of their patients and what reaction is to be expected from them if the state medicine bugaboo should become a universal fact.

"Our patients do not want us to abdicate our position of trust, confidence and independence to become government employes," Robert M. Burns, of St. Paul, declared to members of the Red River Valley Medical Society at Crookston recently. "They would rather, far rather, bear the hardships of private medical costs than accept state medicine.

"While we have gained much knowledge from foreign countries we cannot accept their system of practice. It would be a misfit here, a fatal departure from American traditions. Our constitution guarantees to all the blessings of liberty."

Dr. Burns also pointed out that state medicine has very little to offer that will solve the difficult problem of the costs of medical care.

"We have full respect for those of our profession who are already doing work that might be considered a sample of state medicine. They have chosen their line of work for one reason or another, according to their adaptability. It is largely a matter of administration and enforcement. They are an essential group to the medical profession but their work is entirely different from the private practice of medicine. It is unthinkable as well as undesirable that we should all follow their example."

PRESIDENT'S LETTER

THERE was a time within the memory of all of us when physicians banded themselves together only for the advancement of science. Their officers were invariably men of conspicuous scientific achievement.

Last July, the Minnesota State Medical Association occupied itself during several important sessions of the sixty-second annual meeting with such unprecedented matters as state and national legislation affecting the profession, economics, veterans' relief, cost of medical care and the public relations of the physician. At that session the state association also elected a country doctor for its president.

I find these very interesting and important commentaries on modern medical practise. That is partly, perhaps, because I happen to be the country doctor chosen. Partly it is because they demonstrate the evolution from purely scientific medicine to social medicine which has come about in America in the last twenty years.

This striking evolution is going on in all of the learned professions but in none so conspicuously as in medicine. It is the inevitable sequel to the striking economic and social shifts of the times.

The doctor has discovered of late that it is not enough to write learned papers to read to his confreres or to distinguish himself in his practise. He has been obliged to form an offensive-defensive alliance with his confreres for his protection as well as the protection of the public. He must battle against the cultist and the fake healer, invested as these latter are with all of the power and glory of modern high power advertising. Upon him, he has found, rests the burden of solution of those baffling problems involved in the high costs of medical care, problems that are inextricably tangled up with all of the economic anomalies and perplexities of modern American life. He must forthwith fortify himself and his profession against the socialistic and bureaucratic tendencies of the times, lest the freedom and integrity and essential ideals of medicine be lost.

It goes without saying that the representatives of this new phase of organized medicine must be men of wide contacts and practical knowledge of organization. It is no longer incompatible with the dignity of the profession and of its glorious traditions that the man chosen to head it during 1931 should not be its most illustrious physician.

For myself, however, I can think of no adequate reason why the great state organization of physicians in Minnesota should have chosen me for the job of president in 1931. I cannot promise to follow with any distinction in the footsteps of my predecessors. I cannot promise more than to conduct myself and the business of this organization as worthily and honestly and energetically as I am able.

But I have had, as it happens, a considerable experience unofficially, with Minnesota politics and Minnesota legislators, and I can promise, knowing the men who have consented to serve on the Legislative Committee of the State Association, that the vitally important legislative program of our organization will be carried on effectively this year.

In fact, I ask every member of the Association to read over the membership of all our committees. It should inspire confidence in the whole association to note again the high type and quality of men willing to work for the good of the medical profession.

Fortified by this confidence, I start now upon one of the most important years and undertakings of my life. I pledge my fellow members of the Minnesota State Medical Association who have placed their trust in me, the utmost of my abilities and energies and strength in their behalf.

L. Sogge

President
Minnesota State Medical Association.

CONSULTATION BUREAU

WM. A. O'BRIEN, M.D., *Director*
Minnesota State Medical Association
11 West Summit Avenue
Saint Paul, Minnesota

1. *Question.*—I have a patient with a definite pernicious anemia who has been on liver treatment for some three years or more. The blood picture is staying up well but for the past four or five months the patient has had a lot of trouble walking. She falls and stumbles very easily, and now cannot walk at all without help. I am wondering if anyone has reported any cases of pernicious anemia where the blood picture has remained practically normal on liver therapy and yet there has been a gradual increase in the cord symptoms. Is there any special type of treatment that will help diseased cord symptoms? She apparently needs no treatment as far as the blood goes.

   Answer.—Early reports regarding improvement in involvement of the nervous system were pessimistic, but more recently they have been more encouraging. There have been several reports which indicated that the patient developed cord changes after the blood had returned to normal. B. M. Baker, Jr., Bordley, J. B., III, and Longcope, W. T., MINNESOTA MEDICINE, XIII:815, (Nov.), 1930, report forty-four cases of pernicious anemia in which there were symptoms referable to change in the central nervous system who have been treated by liver or a combination of liver and liver extract. Of twenty-three cases followed for more than six months, improvement in symptoms and signs occurred in 55.17 per cent. In twenty-one cases treated less than six months improvement was noticed in only 31.25 per cent. In eight cases of outspoken subacute combined degeneration, treated for more than tei months, there was improvement in symptoms and signs in 58.9 per cent. They state that it is essential that large quantities of liver, or liver and liver extract, be employed over long periods of time. They recommend 400 grams of liver or more daily or its equivalent. No results need be expected in less than six to eight months. The longer the patient continues the treatments, the less liable it is for remission to occur.

2. *Question.*—A primipara has engaged me to confine her later on this month. Her urine looked bloody on examination and I found it loaded with albumin. I had her wash off the parts thoroughly before collecting the same so as to avoid contamination. Can you outline a course of treatment? What is the prognosis for mother and baby?

   Answer.—The dark colored urine is either due to blood or concentration. It would be advisable to do a microscopic examination of the sediment before deciding which was the cause. Albuminuria in a pregnant woman may mean toxemia of pregnancy. It is advisable to make blood pressure examinations although toxemia might be present without elevation of the blood pressure. Usually edema can be found. Non-renal causes of albuminuria are cystitis, pyelitis and contamination from the external genitalia. While looking for red blood cells, it would also be advisable to look for pus cells. If you make a diagnosis of toxemia of pregnancy, the patient should be put to bed, the intake and output watched, and a restricted protein diet given. It is also advisable to increase the carbohydrate in the diet and to make frequent observations of the blood pressure. The fetus may be dead at this time. The prognosis for the mother in toxemia pregnancy is usually good but not so good for the fetus. You had better examine the urine and if only albumin and casts are present, treat the patient as a potential toxemia case.

3. *Question.*—A married man, while working with a thresher got a barley beard in the lower lid of his left eye. The beard pierced the lid. The eye pained him so intensely that he was taken in for examination and treatment. He arrived two hours after the accident. A portion of a barley beard was removed. The pain was so great, however, through the night that he returned for a second examination and treatment, and more of the beard was found and taken out. He did not obtain relief. The following day the pain was more severe and examination revealed an acute left frontal sinusitis. What is the etiology of the frontal sinusitis? Could this be secondary to an infection in the eye? If so, by what route? Was it through the duct up the nose? Through the lymphatics or through the blood stream? Is the sinus infection entirely incidental?

   Answer.—It is possible for a conjunctivitis to invade the sac of the lacrymal duct and go up through the nose to the frontal sinus. This is rather remote, however, and the acute frontal sinusitis is probably an incidental affair. If it did occur, it would probably be through the duct and not through the blood stream or lymphatics.

4. *Question.*—A nine year old girl turned her right ankle. X-ray the next day showed the fibular epiphysis a trifle wider than the tibial and the toes swung in. I put it up straight. There has been no pain and she can extend the foot and swing it to both sides. The swelling is almost gone (26 days) and almost no tenderness can be elicited on manipulation. But ecchymosis is still present and apparently not fading below the tibia. Except for this, I would think that everything was fine, but fear non-union on account of the delay in absorption of the blood.

   Answer.—Your patient apparently had an epiphyseal separation and was well treated. The almost complete disappearance of swelling, absence of tenderness on massage and manipulation, and active motion of the foot indicate that she is making satisfactory progress. The fact that ecchymosis still persists is probably independent of the injury to the bone. Conversion of blood into pigment and absorption may be delayed under circumstances over which we have no control. If she does not make satisfactory progress, please let us know, but the opinion expressed was that she was doing all right and that the blood would probably clear up within the next few weeks. As long as two months may elapse before the blood disappears, according to the literature.

5. *Question.*—Please give me the treatment for inevitable abortion.

(Continued on page 108)

REPORTS AND ANNOUNCEMENTS OF SOCIETIES

MEDICAL BROADCAST FOR THE MONTH

The Minnesota State Medical Association Morning Health Service

The Minnesota State Medical Association broadcasts weekly at 11:15 o'clock every Wednesday morning over Station WCCO, Minneapolis and Saint Paul (810 kilocycles or 370.2 meters).

Speaker: William A. O'Brien, M. D., Associate Professor of Pathology and Preventive Medicine, Medical School, University of Minnesota.

The program for the month of January will be as follows:

January 7—Carbon monoxide poisoning.
January 14—Peanut bronchitis.
January 21—Cause of the common cold.
January 28—Cancer of the breast.

A. M. A. EXHIBITS

Applications for space in the Scientific Exhibit at the next annual meeting of the A. M. A. to be held at Philadelphia next June, should be made to Thomas G. Hull, Director of the Scientific Exhibit, care of A. M. A. Completed application forms should be in the hands of the director by February 20.

1931 STATE MEETING

Three major symposia on Infant and Maternal Welfare, Diagnosis and Therapy, were proposed for the scientific program of the seventy-eighth annual meeting of the Minnesota State Medical Association by the Committee on Scientific Assembly at its first meeting in St. Paul, Saturday, December 13, 1930.

The annual meeting will be held May 4, 5 and 6, at the Nicollet Hotel in Minneapolis with a morning session at the University Hospitals and Medical School.

Members of the Committee present were A. E. Hedback, W. H. Condit, J. A. Watson, E. L. Regnier, W. A. O'Brien, of Minneapolis, Harry Oerting, E. A. Meyerding, St. Paul, P. G. Boman, Duluth, and A. W. Adson, Rochester. Their tentative arrangements for the May meeting provide for the Council meeting, Monday afternoon, May 4, and the House of Delegates Monday evening. Tuesday morning is to be devoted to clinics at the University Hospitals, Tuesday afternoon to the symposium on Infant and Maternal Welfare. The symposia on Diagnosis and Therapy are to occupy Wednesday morning and afternoon sessions respectively.

The annual banquet of the organization is scheduled for Tuesday evening at the hotel.

Dr. Ray Lyman Wilbur, secretary of the White House Conference on Child Health, will be invited to speak at the banquet. Dr. A. B. Kanavel, Chicago, professor of Surgery at Northwestern University and president of the American College of Surgeons, is expected to attend as one of a number of distinguished guests and speakers.

Invitations will be sent out to the entire membership to make further suggestions as to the program and to participate with papers. The next meeting of the committee will be January 24, 1931, at the Nicollet Hotel, Minneapolis.

RAMSEY COUNTY MEDICAL SOCIETY

At the meeting of the Ramsey County Medical Society, Saint Paul, November 24, 1930, Dr. O. W. Holcomb was elected president, Dr. E. V. Goltz, vice president, and Dr. A. A. Schulze, secretary-treasurer (re-elected). Dr. Robert Earl was re-elected Trustee of the Boeckmann Library-Building Fund. Dr. Gordon R. Kamman presented a report of a case of "Alcoholic Polyneuritis with Cranial Nerve Involvement—Korsakow Syndrome" and Dr. Edward Schons gave a paper on "The Significance of Gas Distention in the Diagnosis of Small Intestinal Obstruction and Stasis (illustrated)."

WASHINGTON COUNTY MEDICAL SOCIETY

Washington County Medical Society held their monthly meeting November 11, 1930, at the Stillwater club rooms.

Dr. Emil S. T. Geist, of Minneapolis, the guest essayist for the evening, presented five reels of the original Bohler fracture moving pictures.

If you can visualize a dozen fellows close together in a dark room with only the pictures visible, and Dr. Geist to explain every important point, you can understand how enjoyable those privileged two hours were.

E. Sydney Boleyn, *Secretary.*

SCOTT-CARVER COUNTY SOCIETY

Lieutenant Governor-Elect Henry Arens addressed the regular December meeting of the Scott-Carver County Medical Society at Jordan, December 9, on "Medical Legislation." H. M. Johnson, chairman of the Legislative Committee of the Minnesota State Medical Association, and Mr. F. Manley Brist, investigating attorney for the Minnesota State Board of Medical Examiners, outlined the work of their respective organizations in initiating and enforcing the Basic Science law. Dr. I. J. Murphy, Minneapolis, read a paper on "Radium Therapy."

The next meeting of the society will be held at Chaska the second Tuesday in February.

RED RIVER VALLEY SOCIETY

E. A. Meyerding, secretary of the State Association, and R. M. Burns, St. Paul, were speakers at the annual meeting of the Red River Valley Medical Society, December 9, at Crookston. Dr. Meyerding talked on "Public Relations," and Dr. Burns on "The Trend of Medicine."

Henry Edstrom, Crookston, read a case report on "Myelogenous Leukemia."

The meeting was preceded by a banquet at the Hotel Crookston, to which members of the Ladies' Auxiliary of the society and others were invited.

NICOLLET-LE SUEUR COUNTY SOCIETY

The Nicollet-Le Sueur County Medical Society and the Le Sueur Commercial Club held a joint meeting at the Le Sueur club rooms, December 8, 1930. Mr. F. Manley Brist, attorney for the State Board of Medical Examiners, spoke on medical legislation and its enforcement.

CAMP RELEASE DISTRICT SOCIETY

At the annual meeting of the Camp Release District Medical Society held December 4, E. A. Meyerding, St. Paul, and S. H. Boyer, Duluth, president of the Association, were speakers.

Gregor Schoofs, North Branch, is the new secretary of the Chisago-Pine County Medical Society.

OLMSTED COUNTY SOCIETY

D. M. Berkman, Rochester, was elected president of the Olmsted County Medical Society at the Annual Meeting, December 9, at Rochester. Other officers for 1931 are A. B. Evarts, vice president, and M. C. Piper, secretary, both of Rochester.

WATONWAN COUNTY SOCIETY

New officers of the Watonwan County Medical Society are: W. J. McCarthy, Madelia, president; H. B. Grimes, Madelia, secretary-treasurer; F. L. Bregel, St. James, censor and delegate. The legislative committee for the society includes: O. E. Hagen, Butterfield; Albert Thompson, St. James; and H. B. Grimes, Madelia.

RANGE MEDICAL SOCIETY

The newly organized Range Medical Society, organized under the St. Louis County Medical Society, met Thursday, December 4, 1930, at Virginia. Dinner at the Fay Hotel was followed by a scientific meeting at the City Hall. B. S. Adams, Hibbing, addressed the latter meeting on "Fractures of the Fingers." C. B. Lenont, Virginia, talked on "Blood Transfusion," and C. W. More, Eveleth, on the American College of Surgeons meeting.

Officers of the new society for 1931 are: H. E. Binet, Grand Rapids, president; C. W. More, Eveleth, vice president; C. W. Bray, Biwabik, second vice president; Fred McK. Ruby, Hibbing, secretary.

RICE COUNTY SOCIETY

E. C. Rosenow, Rochester, addressed the annual meeting of the Rice County Medical Society at Northfield, December 11, 1930, on "Poliomyelitis." The following officers were elected: President, J. M. Murdoch, Faribault; first vice president, W. E. Wilson, Northfield; second vice president, Edmond Backe, Kenyon.

Dr. A. M. Hanson, Faribault, was elected delegate, with F. J. Lexa, Lonsdale, alternate.

Censors for the year are: W. H. Rumpf, Faribault; F. M. Babcock, Northfield; M. L. Mayland, Faribault.

The following members were appointed to act on the Publicity Committee: F. M. Huxley, Faribault; P. A. Smith, Faribault; O. P. Thorson, Faribault.

WOMEN'S AUXILIARY
Minnesota State Medical Association

President—Mrs. S. S. Hesselgrave, St. Paul
Chairman Press and Publicity—Mrs. E. A. Meyerding, St. Paul
Editor—Mrs. A. A. Passer, Olivia

The Executive Board of the Women's Auxiliary of the State Medical Association held a regular meeting at the Lowry Hotel, St. Paul, the third of December. Nineteen members attended the session, which was addressed by Dr. E. A. Meyerding of St. Paul. Dr. Meyerding gave an illustrated talk on "The Minnesota Public Health Association and Its Co-relation to the State Medical Association." Mrs. S. S. Hesselgrave, president of the State Auxiliary, gave an interesting report of the meeting of the National Executive Board, which was held in Chicago the fourteenth of November.

The January meeting of the State Executive Board will be of a "legislative" nature. The Legislature will then be in session and the Women's Auxiliary will learn from the State Medical Association what will be expected of it in regard to legislative measures.

CAMP RELEASE AUXILIARY

Camp Release Auxiliary held its annual meeting at Granite Falls the fourth of December. Mrs. A. A. Passer, retiring president, presided at the business session, which was followed by a joint banquet with the Camp Release Medical Society. Speakers at the banquet were Dr. Boyer of Duluth and Dr. Meyerding of St. Paul. Dr. Boyer talked on "Organization," and Dr. Meyerding stressed the work of the Women's Auxiliary and gave that organization many helpful suggestions as to future usefulness. Camp Release Auxiliary has a membership of twenty-six, but due to the fact that the membership is widely scattered the activities have been mainly social. However, there is a growing interest in serious work, without which no auxiliary can long survive. The following new officers were elected: President, Mrs. J. S. Kilbride, Canby; first vice president, Mrs. M. C. Johnson, Dawson; second vice president, Mrs. G. H. Mesker, Olivia; secretary, Mrs. R. C. Adams, Bird Island; treasurer, Mrs. Leon G. Smith, Montevideo.

RAMSEY COUNTY AUXILIARY

The Women's Auxiliary of the Ramsey County Medical Society has issued an attractive Year Book containing the programs for the season. The December meeting was held at the home of Mrs. H. E. Hunt, 1490 Summit Avenue, Saint Paul. A talk on her recent trip to Russia was given by Mrs. Walter E. Ramsey. Following the talk tea was served with Mrs. E. M. Hammes and Mrs. A. G. Schulze presiding at the tea table.

Mrs. H. O. Skinner, 2190 Princeton Avenue, Saint Paul, will be hostess to the Auxiliary in January, and the March meeting will be held at the home of Mrs. Walter D. Brodie, 665 Montcalm Place. The activities of the Ramsey Auxiliary will be concluded in May

with a picnic at the summer home of Mrs. Henry N. Klein, at Turtle Lake.

Mrs. E. M. Hammes is president of the organization. Committee chairmen appointed by Mrs. Hammes for the year are Mrs. C. K. Williams, philanthropy; Mrs. Karl Dedolph, program; Mrs. H. E. Binger, hospitality; Mrs. Philomen Roy, membership; Mrs. W. H. von der Weyer, ways and means; Mrs. J. Richards Aurelius, telephone; Mrs. H. O. Skinner, publicity; and Mrs. M. Keller Knauff, year book.

HENNEPIN COUNTY AUXILIARY

The Women's Auxiliary of the Hennepin County Medical Society held its annual Christmas party December 17 in the library of the society in the Medical Arts building, Minneapolis. Christmas carols were sung by a group of the members under the direction of Mrs. C. O. Maland. Two of the artist members of the auxiliary, Mrs. E. T. W. Boquist, soprano, and Mrs. Ivar Sivertsen, reader, gave the program. After the program a Christmas tea was served, arrangements for which were made by Mrs. Emil Geist, with Mrs. J. M. Lajoie as co-chairman.

We quote from the American Medical Association Bulletin the following ten points by Dr. H. W. Salus of Pennsylvania concerning the worth of such an organization as the auxiliary:

1. The Pennsylvania Auxiliary has made more progress in six years than the men's society in sixty.
2. The doctor used to bring his wife to conventions. Now the wife brings the doctor.
3. The auxiliary has added polish and refinement to these gatherings.
4. It has created a better bond of fellowship among the men and a lasting friendship among the women.
5. It has permitted the woman to become an integral part of the life of the man, socially, professionally and commercially.
6. It has spruced up the appearance of the men.
7. It has interested the local merchant and brought him business.
8. It has given the doctor much-needed rest and recreation in the midst of his serious scientific meetings.
9. It has brought daughters into more intimate relations with the medical interests of the family life.
10. It has stimulated the medical society not to be outdone in thought and activity.

HORMONE AND CANCER

The effort to influence the growth of cancer by various organ extracts is being widely made at present. Logically it might be profitable to inquire why such trials are being made, since every pathologist sees cancer growing freely in those organs whose hormonic activity is high. The work of Bishoff and his colleagues should be repeated with a tumor that does not spontaneously recede and, if the results are the same, should close the discussion on the possible therapeutic action of extracts from the group of organs tested. (Jour. A. M. A., November 1, 1930, p. 1350.)

PROCEEDINGS MINNESOTA ACADEMY OF MEDICINE

Meeting of November 12, 1930

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, November 12, 1930. Dinner was served at 7 o'clock and the meeting was called to order at 8 o'clock by the President, Dr. E. S. Geist.

There were 43 members and 2 visitors present.

Minutes of the October meeting were read and approved.

The scientific program of the evening consisted of two theses and four case reports, as follows:

Dr. Alexander Stewart (St. Paul) read his Thesis entitled "Observations on Serotherapy in Scarlet Fever." (To appear in Minnesota Medicine at a later date.)

DISCUSSIONS

Dr. W. R. Ramsey (St. Paul): I think Dr. Stewart has covered the field very well. The effect of the serum antitoxin in the severe cases is very marked; as he said, it is sometimes spectacular. The disease is so treacherous that one is warranted in giving the serum, and now that the dosage is so small, that is, so concentrated, one is not likely to get serum sickness. The matter of prophylaxis is still very much in doubt. Parke, Zingher, and others have come to the conclusion that this is still in the experimental stage and have given no definite conclusions, but they hope to have collected enough cases and data to have something definite in the next year or two. Some of these cases do get a terrific reaction.

Dr. Frank Wright (Minneapolis): My experience with scarlet fever is limited to one case in which I was particularly interested. Eight years ago this child, then five years old, developed scarlet fever. She had a temperature of 104° and two nurses constantly for five weeks. She developed an adenitis—both sides of her neck—and a double otitis media and apparently was going to die. As a last resort it was decided to give her some foreign protein therapy. Not being able to find horse serum in the Twin Cities at the time, she was given diphtheria antitoxin. Her temperature had been 104°, and after this first dose it dropped to 102° in six hours. After the second dose her temperature dropped to normal and stayed normal, and in two weeks she was able to be taken south. Now whether or not it was due to diphtheria antitoxin, or the use of foreign protein, or whatnot, she got well.

Dr. H. T. Nippert (St. Paul): I should like to ask Dr. Stewart how he gives the serum, whether intramuscularly, intravenously, or subcutaneously; in very toxic cases would he advise the intravenous use?

With reference to Dr. Wright's remarks, I might add that before scarlet fever antitoxin came into use we had sometimes used diphtheria antitoxin and got fairly good results. We also used antistreptococcic serum and thought we got fairly good results with that too.

Dr. Stewart (in closing): Answering Dr. Nippert's

question as to the method of administration, we give the serum intramuscularly except in some of the late cases we give it intravenously. The first serum that came out we gave intravenously. I recall one case which I tried giving it intramuscularly, before the serum was made up in the concentrated form. It took me about an hour and a half to give that much. We formerly had a lot of trouble and quite a lot of reaction.

In connection with Dr. Wright's remarks on the use of diphtheria antitoxin, we have used that a number of times and thought we got good results, but we get better results from the specific serum.

Dr. William H. Hengstler (St. Paul) read his Thesis on "Neurological Aspect of Head Injuries."

DISCUSSIONS

Dr. A. S. Hamilton (Minneapolis): I have listened to this discussion with great interest and think Dr. Hengstler has covered the subject fully, clearly and conservatively. It is certain that the number of head injuries one sees has increased in recent years; at least those that are classified in reports under the heading of fractures of the skull are so increased. No doubt the coming of the automobile and the speed at which it is driven is to blame for much of this increase, but the *x*-ray also is responsible for many of these reports. At least, it seems to me that what we now call severe fractures of the skull were in my early days called fractures; and those cases which we now see, where one physician sees a line and calls it a fracture and another physician, equally skillful and equally honest, sees the same case and doubts the presence of fracture, were all called concussion without question.

Of course, the *x*-ray has done a great deal to aid us in diagnosis but it is not the fracture of the bone which determines the severity of the injury; it is the degree of involvement of the brain covering, of the blood vessels, of the cranial nerves, and of the brain tissue itself, which determines the severity of the head injury, and here the *x*-ray does not help us greatly. In determining the severity of the injury, one must give close attention to the degree and duration of the unconsciousness. If the patient is in a condition of coma or even of profound stupor and remains so for two or three hours or more, it seems to me that we must assume something more than an ordinary concussion. Of course, in a world where nearly everyone seems to be insured, it is often difficult to get the exact facts. As an illustration of this, I was yesterday looking over two records of the same man. One was given by the man himself in which he recorded a statement of absolute unconsciousness for three hours following his injury. The other report, made by the man who assisted him, stated that he went for help directly after the accident and returned in ten minutes. The patient was sitting up and was conscious. With assistance, he got into an automobile and was driven several miles to a hospital. He was an Irishman and in the course of his journey, he laughed and joked, remarked that you could not kill an Irishman, smoked a cigar and was finally delivered in a conscious condition at the door of the hospital. This does not prove that the man was lying, for, in head injuries, all incidents are sometimes blotted out of the memory for a certain time so that the patient cannot go back and reconstruct his life. Certainly, however, the case where the man responds in a clear-headed way, even though he cannot afterward recall what happened, is very different from that one in which the individual is in a profound coma for a considerable period. Where there is loss of the deep reflexes and marked pupillary changes, including failure of the pupils to respond to stimuli, the case is certainly serious.

Unfortunately, it is very difficult at the present time to find any statistics which would be generally accepted as to what the results are likely to be in the average case of head injury. As I have already remarked, we are so generally insured against injury and the desire for a little compensation is so widespread that a motivating psychologic factor is very likely to develop symptoms which at best are often largely of a subjective character and keep alive these distressing phenomena which, without the compensation situation, would certainly long ago have disappeared.

Dr. Hengstler has referred to epilepsy following head injuries. Unfortunately, here again, we have no reliable statistics on which we may depend to determine the likelihood of epilepsy in any given case. I have seen statistics where epilepsy is said to have developed in cases of head injury with fracture of the skull in as high as thirty per cent of cases. Judged by my own experience, such a statement is ridiculous unless the author is dealing with a specially selected series of very grave cases. Like the author, as I understand him, I look upon epilepsy as a situation where there must be some fundamental constitutional condition and if trauma is to be looked upon as a cause of epilepsy, if the above opinion is correct, it must be looked upon only as a precipitating or exciting cause and not as the primary cause. I have, however, seen cases of convulsions of generalized character developing in cases following head injury where I can hardly assume the relationship was merely coincidental and I have felt that sometimes injury to the brain may bring on generalized convulsions.

What is known as delayed hemorrhage constitutes an interesting feature in a very limited percentage of head injuries but a sufficient number of cases have now been reported so that one cannot doubt their existence. The period between the time of injury and the onset of the cerebral hemorrhage may be anywhere from a day or two up to as long as a few weeks and it is worthy of note that in the cases of delayed hemorrhage reported there has frequently been found evidence of pathological conditions in the vessels so that the injury would have been looked upon ordinarily as only a precipitating cause.

Dr. J. F. Corbett (Minneapolis): I enjoyed this essay a great deal. As the paper was read, there went before my mind the positive indications for operation. First and foremost is the case where progressive symptoms develop, where there is a little bleeding from a vessel outside of the dura. The patient may develop paralysis, aphasia, unconsciousness, and finally reach

the stage of medullary compression and die. These cases are common causes of death in head injuries. In the statistics of Le Cont in Cook County, a major part of the deaths from head injuries were attributed to extradural hemorrhage. The cure is operation. The operation is in simply removing the clot, if necessary the ligation of the vessel, and the outcome is pretty fairly good. I have had an opportunity to do this a little over thirty times and have had two deaths, and both of these were cases that reached the stage of medullary compression. Another indication for immediate operation is where there is a compound fracture. Thorough immediate débridement is imperative.

But there is another condition which demands immediate operation and that is a fracture extending into the sinus into the frontal bone. I recently had a case where an abscess developed into the frontal region that came from such a crack being neglected. Depressions are commonly corrected by elevation or removal of the fragment.

The spinal puncture needle is very valuable and I had certain rules at the General Hospital to follow in the use of spinal punctures, but every once in a while somebody would go wrong and use the spinal puncture where it should not have been used. There are cases where it should not be used. First, if your patient is in shock, do not use the spinal puncture. Second, if there is threatened embarrassment of the medullary centers, do not use spinal puncture. And, third, although it is difficult to lay down exact rules for diagnosis, where there is bleeding in a large vessel in the brain or skull you may get a fatal ending from using spinal puncture; but I do not know how you can anticipate that in advance.

Dr. Herbert Jones (Minneapolis): It seems to me this paper is very opportune at this time. We formerly had the injuries caused by horses and those were direct accidents. Then we passed into an era of railroad accidents in which the large element was that of shock. The saying among the old railroad surgeons was that the injuries were so severe that the tissues were shocked and the patient did not do well. These accidents have been largely overcome by the railroads. We are passing now into the age of automobile injuries. When an automobile hits a person that person is temporarily unable to control his head; that is, the head hits the ground or some object and we get the peculiar head injuries that we see in ever-increasing numbers. Those accidents are especially in the neurologist's field to deal with, as there must be a competent diagnosis in order to decide whether the person is so shocked that the outcome will be fatal if operated. A certain number must be operated upon in order to prevent devastating hemorrhage into the brain tissue. And the decision is a very important one. This paper is a very timely one.

Dr. J. C. McKinley (Minneapolis): Martland published an article on "Punch Drunk" in the Journal of the A. M. A. in October, 1928, which seems to me to bring up a point which is not appreciated in our consideration of concussion and contusion of the brain. He calls attention to the high incidence of commonly progressive neurologic syndromes (ataxia, tremors, Parkinsonian states) in prize fighters, especially those of the rough-and-tumble type of poor boxer who takes much head punishment over a considerable period of time. He thinks that the symptoms are due to petechial hemorrhages in the brain. Now we usually think of the knockout in a fight as a pure case of concussion without permanent injury, but obviously from Martland's observations a small amount of permanent damage is done to the brain with each knockout blow. The effects are cumulative until a full-blown neurologic picture develops. This indicates the difficulty, if not impossibility, of separating concussion, with its temporary effect of loss of consciousness, from contusion, with its permanent anatomical and often functional damage. One should be guarded in a given case in speaking of a concussion without contusion, as it is doubtful if such a circumstance actually occurs.

Dr. A. W. Ide (St. Paul): In our experience in railway work, head injuries have been becoming more and more infrequent. The few injuries that we do see are not caused by accidents incident to the operation of trains. Occasionally some one is injured as a result of a speeder accident. These speeders are small gasoline inspection cars. In spite of every precaution, the light cars sometimes leave the rails and throw the passenger from his seat.

It seems to me that one of the greatest problems in connection with head injuries is getting the men back on the job. Headache and dizziness persist and are very annoying. Patients are apt to be uncertain of themselves. They doubt their ability to continue work over prolonged periods.

A sympathetic attitude on the part of the physician is very necessary. These patients need encouragement.

Dr. Hengstler (in closing): I appreciate very much the discussion of this paper and wish to thank the gentlemen for it.

I want to say that I agree most heartily with what Dr. Corbett said about the use of spinal puncture, believing that spinal puncture should be used, but used with discrimination; and I agree with him that in cases of free intracranial hemorrhage, its outcome cannot be foreseen.

With respect to epilepsy, my experience with this following head injuries is somewhat limited. The cases that I have had have all been in connection with litigation and the patient was claiming epilepsy as a result of the head injury. Some claimed types of grand mal and some of petit mal, but in each instance investigation proved these individuals came from epileptic families where the history had been covered up. One can conceive of an individual who had epileptic parents and who has an epilepsy in abeyance, and then who experiences an accident which lights up this epilepsy. That, I think, might be admitted, but in my cases where they had true epilepsy following head injury, it has always been of the Jacksonian type. I think my experience has been too small, perhaps, to make a definite statement about the matter of epilepsy. In the cases brought into court, there is no question but what a thorough investigation should be made of the family

and antecedents to prevent concealing a history of previous epilepsy.

Since writing this paper, and within the last two months, I have helped take care of a woman who was injured in St. Paul, and who was in a state of unconsciousness for fifty-two days, and finally died of pneumonia. She was brought into the hospital with a respiratory rate of four per minute, and a pulse in the fifties, and in profound shock. We decided on a spinal puncture and drained off 60 c.c. of bloody fluid. She improved following the first puncture and we continued these. After some days of bloody fluid, it began to clear, going through the characteristic changes, and the woman seemed on the road to recovery. She never got beyond a certain point, however. Her eyes were open and she would take food, but she knew nothing of what was going on around her. Those of us who were seeing the patient right along advised against opening the head, but her husband had friends who were very insistent upon it. We did not yield, however, and at autopsy our judgment was proven correct. The skull was opened and she had no clots of any kind, intracranially, but the right cortex was very badly lacerated, and had she lived, of course, she would never have regained normal mentality. In this case we were fortunate, at autopsy, in getting the evidence which proved that the proper therapy had been used.

DR. H. P. RITCHIE (St. Paul) gave the following three case reports:

1. A Case of Prenatal Sarcoma.
2. Multiple Congenital Clefts of the Face.
3. A Transplant of the Female Breast.

I have three cases which I believe are worthy of our permanent records.

Case 1 is that of a baby born on June 20, 1929, at Pine City, Minnesota, under the care of Dr. Stratte. There is nothing in the family history that would have any bearing on the case. The carrying period was normal; the confinement was normal. Subsequent to birth, Dr. Stratte remembered that the amniotic fluid as it was evacuated was distinctly bloody, but this was not noted at the time for obvious reasons. The baby was born white with a large outgrowing spongy, bleeding tumor which involved the right heel and foot. A tourniquet was placed on the leg and as soon as the mother was cared for the baby was transported to the Children's Hospital in St. Paul, where it arrived about two hours after birth. Dr. Ray Shannon saw the case, appreciated the critical situation and transfused with the father's blood. Blood count, hemoglobin 48, reds 1,790,000, blood grouping IV. I was called in to pass upon the possibility of surgical procedures other than amputation. The indication for this operation appeared imperative. After another transfusion, the leg was amputated in the lower third at the fifth hour of the baby's life. The specimen was given to Dr. Kano Ikeda, who made many sections of this tumor. Figure 1b is a photomicrograph of but one of several sections made. It shows a densely cellular tumor with nerve fibers, greatly enlarged, running throughout. Dr. Ikeda's report, copied from the history, is as follows:

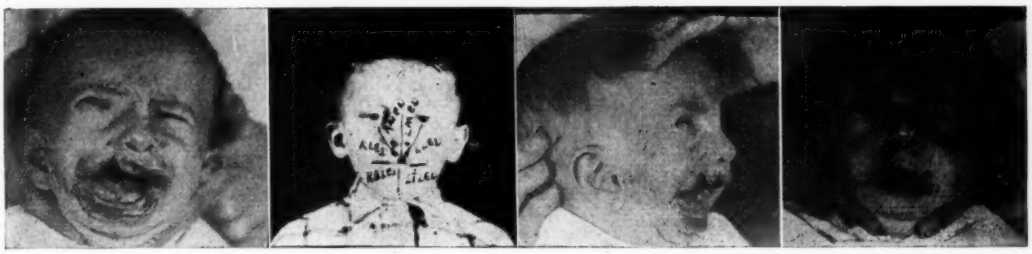
a b c d

Fig. 1

"A soft fungoid mass about 6 cm. in diameter protrudes out of the tibial aspect of the right foot and along the inner surface of the ankle. The surface of the mass is rounded and devoid of skin. The plantar aspect of the mass is irregular and broken. The cut surface shows a soft whitish cellular tissue with areas of hemorrhage. The tumor tissue infiltrates diffusely through the soft tissue of the tarsal portion of the foot. The aspect of the os calcis appears broken through by the tumor growth, while a cordlike trunk 1 cm. in diameter is present along the course of the tendo achillis. The tumor is composed of compactly arranged bundles of large pale staining spindle cells running in all directions. Many mitotic figures are noted. Diagnosis: Spindle-cell sarcoma."

Examination of the gross specimen showed that the amputation was through or so near to the tumor that, should the baby live, recurrence would surely follow. The tumor had extended, pushing aside the neighboring tissue alongside the tendon of Achilles up into the leg. The father was frankly told that the operation was unsatisfactory and incomplete. Re-amputation assumed the aspect of a moral problem. Was it justifiable to further mutilate this infant in the presence of such an active malignant growth? I decided to be influenced by the solution of two questions. Had metastasis already taken place? *X*-ray studies of the lungs were made and were negative. The question of icterus was run down and its presence disproven. The second question was whether the new growth was primarily an osteogenetic tumor? Rightly or wrongly, I have carried in my head the idea that primary bone tumors of the sarcomatous type were eventually lethal. The few cases which have come under the care of Dr. MacLaren, Dr.

Daugherty and myself are all dead, no matter what the point of amputation. The bones of the foot were found to be involved. This question was carefully considered by Dr. Bell and Dr. Ikeda with the conclusion that they were secondarily and not primarily involved. The father was most understanding and coöperative, and I received permisison to re-amputate. The point of amputation received consideration as to whether it should be undertaken in the leg or above the knee. I finally decided that the question was critical and so amputated on the fifth day of the baby's life in the lower third of the femur. The baby was bundled up and taken from the operating table back to its mother in Pine City. Under the care of Dr. Stratte this baby has now developed into a husky beautiful child, fourteen months old at the time of taking the picture in Figure 1d.



a b c d

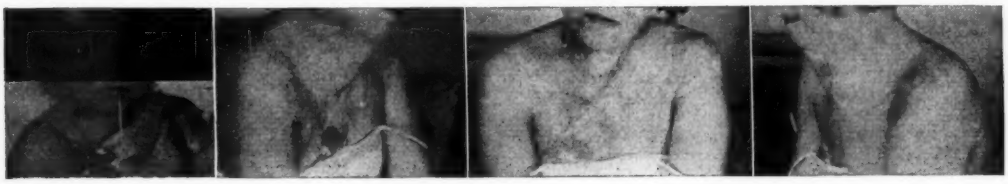
Fig. 2

The value of this case from a surgical standpoint is nothing. These cases must be very rare, but we have not made a complete survey of the literature in this respect. Possibly there are many cases which have not reached the literature. From an embryonal standpoint it is interesting to speculate upon the cause of such a luxuriant growth developing to such a point in utero. It may have some bearing upon the theories of the formation of new growths. I presume from a pathological aspect the proper procedure will be to send all this data to the Sarcoma Commission for further consideration. From a clinical standpoint, the circumstances and problems of this case were as dramatic as in any of my recent experience.

Case 2 is a baby now four months old which shows multiple congenital clefts of the face. In Figure 2a a cleft appears in the right upper lip, commonly described as "harelip"; also a cleft at the right angle of the mouth extending into the right cheek, and a cleft of the left angle of the mouth extending into the left cheek. In Chapter IX, Volume V of Lewis' System of Surgery, I have had the wonderful opportunity to review some of the problems in the field of congenital clefts of the face and jaw. As I read that chapter in cold print I am startled at the controversial tone of it, the indirect and uncertain language of several statements and the over-emphasis on certain parts of the technic. But there is one part which I believe may very well be greatly enlarged. Part II of the chapter is entitled "A Surgical Interpretation of the Embryology." This is the product of a study undertaken through the interested help of Professor R. H. Scammon with a definite purpose to relate the embryology of the upper face and jaw to the surgical problems of this locality. I am in no position to talk embryology. I am still uncertain as to how much of the data recognized as principles of growth is incontrovertibly established; how much of the evidence is due to the direct study in the human embryo; how much is indirect evidence taken from the lower animals and applied to the human embryo. There is, however, a general agreement that the face is formed by the contact, union, or coalescence of separately developing groups of embryonal tissue. In embryological terms these are called "processes." The mandibular processes form the lower jaw, the maxillary processes and the frontal-nasal processes form the upper jaw. In the effort to carry embryology to our clinical problems it seems to me that we should have a broader term of description of the embryonal groups, so as to include all elements, no matter whether it is skin, muscle or mucous membrane, or bone, or whatever kind of tissue, because there is here found all of the superficial tissues of the body and which at some time require surgical attack. In searching for a substitute term, the word "division" was selected as being broad enough to include all elements under consideration for surgical repair. In Figure 2b are marked lines and letters inserted upon a photograph of a bilateral cleft lip. This particular case was selected because the clefts are exactly symmetrical; thus the prolabium of the median embryonal division is beautifully outlined. It is necessary to follow this figure while reading the description.

The lower jaw is formed by the union of the mandibular processes, here described as the right and left inferior lateral embryonal divisions. Should there occur a failure of union of these two divisions, a cleft will result. This cleft must be a median cleft and is here represented by a straight line in the lower lip. The upper jaw has a right and left lateral embryonal division exactly similar to the lower jaw. A failure of union between the right or left superior lateral embryonal division and the right or left inferior lateral embryonal division will result in a cleft at the angle of the mouth, right or left. These clefts likewise must be straight lines because, as in the lower jaw, only two embryonal divisions are concerned in the union. Unlike the lower jaw, the upper jaw has a third division which is called "the median embryonal divi-

sion," inserted in between the right and left laterals. Growth is further complicated by the fact that the division has two points of origin so that the right median embryonal division has a different origin from the left median embryonal division. For instance, the alar cartilage of the right nose has a separate origin and at each angle of the mouth; thus all the lines drawn on the diagram have been clinically demonstrated. Whether or not the surgical interpretation of the embryology can be supported in detail, the statements here made, taken merely as a theory, fit into so many conditions that I believe they are worthy of mature consideration.



a b c d

Fig. 3

growth from that of the alar cartilage of the left nose. So it is perfectly possible to have a failure of proper union between the right and left median embryonal divisions. Should this occur there follows a cleft nose, a cleft columella, a median cleft in the prolabium and pre-maxilla. All of these tissues are derived from the median division. These clefts should be straight-line clefts for the reason that again only two divisions are involved. The common harelip or cleft lip is formed by the failure of the right superior lateral to unite with the right median, producing a right cleft; or the left median to heal with the left superior lateral, producing a left cleft; or both appear in the same case. These clefts are always definitely lateral because they appear to one side or the other of the median division and therefore should be in oblique planes. These latter clefts may also extend into the face and up to involve the lower eyelid on either side or both sides. Every human embryo that starts life may have any one or all of six possible clefts, as shown by noting the lines on the diagram.

A review of this case of multiple clefts reveals that the right superior lateral embryonal division is out of contact with both the inferior and median neighboring divisions. The obicularis of the lip, on the superior right, which stands out definitely as bunched muscle, is entirely out of contact. It has failed to unite with the right inferior muscle and the cleft in the cheek results. It has failed to migrate underneath the skin of the prolabium to come in contact with the muscle which has normally migrated from the left lateral, and a harelip results. There is a suggestion of a cleft continuing from that of the lower lip up through the face to a small but appreciable angle in the lower lid. There is also a dermoid of the right eye.

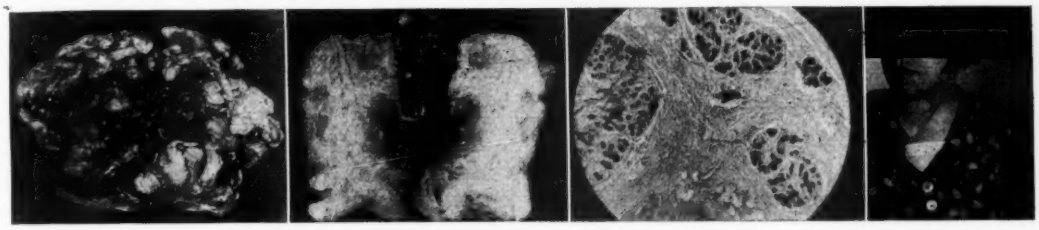
The surgical indications are beautifully outlined and indicate that repair should be made on the muscle principle. In my series I have a cleft nose, I have seen illustrated the cleft columella, I have two perfect median clefts of the prolabium, I have many cases of harelip, both unilateral and bilateral, I have seen illustrated a cleft of the lower lip, and here is a case with the clefts

Case 3 is one to which I call your particular attention. In 1918, a girl five years old was brought to Dr. MacLaren, Dr. Daugherty and myself, suffering from a burn contracture of the neck, of a degree in which the chin was anchored to the manubrium. Two double-ended flaps were outlined—one on the right chest and one on the left shoulder and arm. The distal ends were gradually severed and when freed were placed in the form of a cravat on the denuded area which followed the dissection of the contractures of the neck. There is no picture of the original condition; indeed, one may not have been taken. We were not so careful of pictorial records then as we are today. Figure 3a is a photograph of the girl as she left the hospital and from this it will be seen that the flap from the right chest was placed at the chin and that from the left shoulder at the manubrium. Nothing was heard of the girl except an occasional indirect report that the operations were satisfactory.

On July 15, 1930, the girl with her mother appeared at my office. She had grown into a husky young lady, a junior in high school. She gave a history that everything was well until about four years previously, when the neck began to show gradual and progressive enlargement. Figure 3b is a photograph of the right side showing the very full, rounded neck and also the scar on the right chest. She wore a black band about her neck. When this was removed I was startled to see a well-developed mature nipple in the suture line shown in the front view (Fig. 3c) and left side of the neck (Fig. 3d) in which the nipple is more definitely shown. It is interesting to note that the enlargement had extended underneath the tissue of the left flap, which is very well outlined in this photograph. It took me quite a time to determine the situation. Palpation of the neck revealed a definite organized body under the skin and movable on the deep structures. Evidently in the first operation we were more concerned about the length of the flap and the fitting of it into the raw surface than we were in the tissues contained therein and had unwittingly included the immature infantile breast in the flap and had perfectly encircled the whole nipple

and mammary gland leading into it. When puberty occurred this gland developed normal physiological enlargement.

The surgical problem was first, the question of replacement of the gland to its normal location. I knew of the elaborate procedures necessary to obtain tissue ments of a free graft. The normal bed of this tissue is completely severed and a new field invaded. Anatomical structure must be maintained by the formation of entirely new avenues of nourishment. The margin of failure is enormously widened unless meticulous care is exercised to obtain the most favorable conditions for



a b c d

Fig. 4

to substitute this beautifully placed flap and the time required to do so. The thing that decided me was the question of whether this gland was really a functionating structure and whether it would pursue its normal course should it be replaced. I finally decided to remove it. The next day a half-moon incision was made to remove the nipple, the tissues of the left flap were reflected backwards and a line of cleavage underneath this growth was then determined. At one time during the operation I had the identical picture of an ordinary mastectomy; the only difference being that the base structures were those of the neck instead of the pectorals. The usual care of dissecting the skin to prevent buttonholing and consequent scar was exactly the same. Figure 4a shows the mass removed with the nipple at the lower part of the specimen. Unfortunately this photograph was taken after it had been in a preservative, but it gives the ordinary picture en masse of the normal breast. Figure 4b shows the growth bisected; the size, the contour and the consistency illustrates a familiar picture. Figure 4c is a photomicrograph taken at random showing the regularity of the acini lined with deeply-staining cells and, although the interstitial tissue may be exaggerated, it surely has the picture of a normal breast.

The clinical value of this case, of course, is nothing. I cannot imagine a situation that would require the purposeful transplant of a female breast, but as a case bearing upon some of the questions and principles of tissue transference, it may be of great value. The recent literature upon the subject of transplants indicates that there is a more general recognition of the fact that the free-graft principle of transference is entirely different from that of the flap. The cellular structures of a flap, properly prepared and placed, are always live elements nourished by channels of supply approximately the same as when in their original position and the margin of chance is very broad that these structures will pursue in the new location their normal growth, physiological action and anatomical change, almost exactly similar to their behavior before the surgical attack. Quite contrary consideration must be made of the ele- the formation of new sources of nourishment and the physiological life of these cells revived at the earliest moment or anatomical death results and a failure of the surgical purpose follows. The behavior of the tissues of a free graft, of whatever nature, is by no means settled. There is a great question how many of the original cells revive and follow through their normal growth, how many are substituted by scar tissue, how much of a so-called successful result of a free graft is really due to growth of the original tissue, and how much is due to the possible stimulation of growth of other tissue.

There is one division of this subject, however, where these considerations have been entirely disregarded, *viz.*, the transplant of glands. From a surgical standpoint the claims made for these procedures have exceeded the possibility of tissue transference. Glands have a complicated anatomical structure. They have intricate physiological function, depend for their activity upon direct arterial blood supply, and have a definite system of absorption. We are told that these are all severed and, on the principle of a free graft, these tissues are then placed in another location with the expectation that this complicated anatomical arrangement will be maintained and the physiological action restored. Indeed, it is suggested that the glands be placed in muscles, that the outer surfaces be scarified in the hope of obtaining new channels of nourishment; but when looked at from the principle of free graft and the flap, it seems to me that a gland so transferred can only have temporary effect, must either degenerate or become encysted as a foreign body, and that the clinical results reported following such procedures must be, in the main, psychological.

The mammary gland is the least complicated of any, but if it had been completely severed from its attachment and placed in the neck or elsewhere it would not have given the picture here shown. In this case the mammary gland was at no time severed from its original blood supply. Possibly some part of it may have been cut away, but at all times it has been living tissue, and during the years new channels have had time to

form and consequently when puberty took place we see physiological enlargement. The explanation of its growth in this case appears logical and reasonable and I believe that this gland would have been activated in its odd position by pregnancy.

There is a chance for some one to make complete revision of the whole field based upon the physiology of the cell or groups of cells included in the transplant.

Dr. Arthur W. Ide (St. Paul) reported the following case of Prenatal Sarcoma, and the baby patient was presented before the Academy.

The patient (Baby B.), a girl, is now 35 days old. She was born in the Northern Pacific Hospital, St. Paul, Dr. H. G. Collie attending.

At birth a tumor was seen involving the left leg. This growth measured 16½ cm. in length and 23½ cm. in circumference. It was situated on the anterior surface of the knee and extended upward onto the anterior surface of the thigh and downward on the anterior surface of the leg. There was a small ulcer on the anterior surface of the tumor. General examination showed the baby to be normal except for this tumor. She weighed 7 pounds 12 ounces at birth.

Family History: The parents are both living and are strong and healthy. They have one other child, a boy three and a half years old, who is perfectly normal, well and strong. There is no history of syphilis, rheumatism, gout, tuberculosis, epilepsy or malignant disease The paternal grandmother died of Hodgkin's disease.

The patient has been at home and cared for by the mother except for the first ten days of her life. She now weighs 7 pounds 8 ounces—four ounces less than her weight at birth.

The tumor has not enlarged since birth. The ulcer on the anterior surface of the tumor has extended. It is now 2.5 cm. in diameter.

Diagnosis: The patient was seen by Dr. E. T. Bell, who considered the tumor to be a sarcoma.

It is very probable that the growth is malignant. The *x*-ray indicates that the bone is not the primary seat of the growth. It is firm in consistency. A biopsy so far has not seemed justifiable and for this reason we cannot state definitely the nature of the growth.

The meeting adjourned.

R. T. LaVake, M.D.,
Secretary.

VIOSTEROL

Holtz and Schreiber of Göttingen have reached the conclusion that irradiation of ergosterol produces a number of distinct substances. One is the antirachitic factor, perhaps Vitamin D proper; another is a separate component producing calcification. The two effects, antirachitic and toxic, can be separated; or at least one can be destroyed while the other remains intact. If these observations prove to be correct, they may lead to a desirable purification of viosterol so as to eliminate or diminish the "toxic" features of its action. (Jour. A. M. A., November 22, 1930, p. 1593.)

PROCEEDINGS OF THE MINNEAPOLIS SURGICAL SOCIETY

Meeting of November 6, 1930

Dr. A. T. Mann, President, presiding.

The regular monthly meeting of the Minneapolis Surgical Society was held in the Lounge of the Hennepin County Medical Society, on Thursday evening, November 6, 1930. The meeting was called to order at 8 p. m. by the President, Dr. A. T. Mann. There were 26 members and 6 visitors present.

On account of the length of the program, the reading of the minutes of the October meeting was omitted.

The scientific program was as follows:

Dr. John Rishmiller presented a case report on "Habitual Dislocation of the Shoulder, and the Clearmont Operation," with a general discussion of shoulder joint injuries. (To be published later in the International Jour. of Med. & Surg. and the Surgical Journal.)

Drs. L. H. Fowler and Wm. A. Hanson gave the following report of a case of "Embolic Gangrene of the Foot," the report being read by Dr. Hanson, after which the patient was presented.

The patient was a man aged 54 years, whose past history was essentially negative. On February 4, 1930, he had severe sharp pains in the arms, back and chest, at the level of the nipple line, which were not related to exercise. He had many carious teeth with recession of the gums, gingivitis and pyorrhea. Examination was otherwise negative.

On February 22, 1930, he had six lower teeth extracted, with no reaction following. On May 20, 1930, he had six upper teeth extracted, one surgically, with curettement of the sockets. On May 22, 1930, he began to have sharp pains over the chest of a stabbing character, with clonic contractions of the neck, chest and arm muscles. Six days later he had an increase in temperature to 101°, pulse 95. At this time he complained of pain over the dorsum of the right foot between the large and second toes. There was a small abrasion with an erythema about this area. He had marked hyperesthesia over the dorsum of the foot extending to the outer surface of the ankle. On May 31st he developed a temperature of 103° with a serosanguinous bleb about 3 by 4 inches in diameter over the dorsum and lateral side of the foot. Also a similar bleb 1 by 1 inch appeared over the medial surface of the foot. The whole foot and leg below the knee became markedly swollen, red and indurated. The patient was very toxic, delirious, with a temperature of from 103° to 104° for several days. General supportive measures were instituted. The foot and leg were treated by elevation, hot packs, Ochsner's solution for 24 hours, and then continuous irrigation with Dakin's solution. His general condition gradually improved. The cellulitis of the foot and leg cleared up. The skin and subcutaneous tissue of the dorsum and the lateral surface of the foot sloughed away, exposing the extensor tendons. Eventually, clean healthy granulation tissue appeared over this area.

On June 24, 1930, a full thickness skin graft was done. This was not successful, probably owing to the

impaired blood supply to the part. Later, Thiersch grafts were applied with very satisfactory functional and cosmetic results. The patient has a practically normal foot today.

Careful examinations by medical consultants have failed to disclose any pathology in the heart or lungs. All laboratory examinations, including urine, blood, blood culture, *x*-ray, and electrocardiogram were negative.

The pathological process was apparently due to a blocking of some of the smaller terminal vessels of the dorsalis pedis artery by infective or bacterial emboli originating from infection around the teeth.

DISCUSSION

Dr. L. H. Fowler stated that this was a "rapid-fire" fulminating condition. In February the patient had had no trouble following the extraction of six teeth which were pulled on account of marked infection of the gums around the teeth. Three months later he had six more teeth extracted, and in six days the acute chest condition developed, which was apparently due to showers of small pulmonary emboli. Two days later he developed the lesion on the dorsum of the foot, particularly localized at the base of the great toe. First there were blebs on the skin. Soon the whole foot became edematous, and extensive cellulitis developed extending practically up to the knee. Within four days the whole dorsum of the foot was gangrenous. Eventually the superficial tissues of the lateral side all sloughed off down to the extensor tendons. The skin on the medial side of the foot retained its vitality in spite of the subcutaneous tissues being necrotic and infected.

Dr. Fowler called attention to two or three interesting phases of the case. Surgically, it did not present such a difficult problem except combating the infection. Part of the skin grafts used followed Dr. Wangensteen's method of cutting the Thiersch grafts into small bits and burying them in the granulation tissue. They took, and there is practically no difference today between those and the other areas where whole Thiersch grafts were applied.

Dr. Fowler said that the pathology in this case was interesting: How did these emboli get there? The gangrene was localized to the distribution of the arcuate artery, a terminal branch of the dorsalis pedis, and must have been due to an infective embolic process. He stated further that he had found very little in the literature on this subject; only one case in recent years and that was a case of gangrene of the toes following tonsillectomy and reported in 1929 by Wright, which was associated with a subacute bacterial endocarditis. Dr. Fowler's patient never had any signs of a heart lesion. Pathologists say that very rarely emboli follow operations on the head and neck.

The speaker felt that the only logical explanation for this case just reported was that it was due to a bacteremia coming from the infected teeth, with the bacterial emboli lodging in these terminal vessels and causing the gangrene.

Dr. E. A. Regnier asked how Dr. Fowler accounted for the chest pains?

Dr. Fowler thought this was due to showers of small pulmonary emboli, and added that the patient had had four or five attacks of these sudden severe pains in the chest since he had been at home, but at no time had there been any definite findings in the lungs or in the heart.

Dr. R. C. Webb asked if an electrocardiogram had been taken.

Dr. Hanson stated that an internist had gone over the heart very carefully and there had been *x*-rays and electrocardiograms taken, all of which were negative. He also added that at no time were the toes or sole of the foot involved.

Dr. J. M. Hayes stated that he had been using the so-called Ochsner (or modified Ochsner) method of treating infections of the extremities for many years; and he knew of no one who had ever used it properly, going back to the old method of using plain hot packs for this purpose. Dr. Hayes felt that there was no doubt of its superiority over the use of the ordinary hot pack.

Many years ago Dr. Ochsner had observed this dressing used with saturated boric acid alone and the results were so satisfactory that he was led to do some experimenting with it. He observed that boric acid appeared in the urine after this dressing had been on a few hours. Taking bacteria from the wound before the pack was applied, and injecting them into the peritoneal cavity of a guinea pig, caused the death of the guinea pig in 24 hours. After this dressing had been on for 48 hours, bacteria from the same wound injected into the peritoneal cavity of a guinea pig failed to cause any disturbance.

Dr. Hayes stated that he and his associates had also carried out some of these experiments and obtained the same results. He added that, although the boric acid alone gave good results, he found that the addition of alcohol and phenol added to the efficiency of the solution. He called attention to the fact that several different solutions have been called "Ochsner's solution," and for that reason the one he used was referred to as "modified Ochsner's solution." The solution accredited to Ochsner in some dictionaries would cause a slough in 48 hours, but the "modified Ochsner's solution" would never cause a slough in 48 hours.

Dr. Hayes believed that the reason this treatment had not become more popular was because the technic of applying it was not carefully adhered to, and that this was very essential in order to obtain the desired results. He said that the pack must be large, consisting of a thick layer of gauze over the skin, then a thick layer of good absorbent cotton, and an outside covering of rubber, making the whole dressing as nearly air-tight as possible. This dressing should not be disturbed for 48 hours except for the solution poured on two or three times a day through a small opening in the least dependent portion of the dressing. The solution Dr. Hayes now uses is: boric acid, saturated solution ounces 28, alcohol ounces 4, and phenol drams 1. The dressing must be kept thoroughly saturated with the solution.

Dr. Hayes called attention to the enthusiasm of some

of those who have recently gone out from the University, over this method of treating these wounds, and their satisfaction in getting these patients back to work so much more rapidly than by other methods.

Dr. A. A. Zierold asked if Dr. Fowler's patient had gangrene before or after the application of the Ochsner's solution.

Dr. Hayes replied that there was a thrombosis present and evidence of a slough before the dressing was put on. This dressing will never give a slough in 48 hours.

Dr. Owen Wangensteen stated that Dr. Hayes may be perfectly right about the use of this solution. He called attention to the fact that when Dr. Hayes began injecting varicose veins others were amused over it but that it was now a universally practised procedure. He said that in the treatment of acute infections there are essentially two factors of primary importance: one is absolute rest, and the other increase of heat in the affected part. Nature does this in part by increasing the temperature locally. He wondered if water poured into the dressing as used by Dr. Hayes would not accomplish the same result, and called attention to the fact that a 1 per cent solution of phenol might cause gangrene in the terminal digits. However, he had seen patients treated by this method and had observed some of the good results which Dr. Hayes had obtained. He said he would like to see Dr. Hayes run a control series of these cases at the University Hospital, using hot water in the pack instead of the Ochsner's solution.

Dr. Hayes stated that no doubt the method of applying the dressing was an important factor and that fair results were obtained with boric acid or even with sterile water, but he had tried these many times. When the infection was severe this was not enough. After trying several solutions the one given above was found to be the most efficacious and, if properly applied, will not fail to localize any infection of the extremity in 48 hours. The suggestion of having some beds for this work in the hospital would be a most acceptable proposition.

Dr. William P. Herbst stated that it is an interesting fact that boric acid has been demonstrated in the urine 55 seconds after immersion of the foot in a tub of boric acid solution, even when the skin is unbroken. (Ref: Kahlenberg, L., Jour. Biol. Chem., 1924-25, lxii, 149, p. 405.)

(The patient was then shown and attention called to the location of the grafts and the excellent result obtained in this case.)

Dr. Thos. J. Kinsella of Glen Lake then read his inaugural thesis entitled "Phrenicotomy for Pulmonary Tuberculosis." This was illustrated with numerous lantern slides showing the condition of the lungs before and after operation.

An Eastman film on "Prostatic Hypertrophy" was shown after the reading of the papers. This proved to be an enjoyable feature of the scientific program and it is hoped that arrangements may be made to have similar films shown at future meetings of the Society.

The meeting adjourned.

H. O. McPheeters, M.D., *Secretary.*

PROCEEDINGS OF THE MINNEAPOLIS CLINICAL CLUB

Meeting of October 9, 1930

The regular meeting of the Minneapolis Clinical Club was held in the Hennepin County Medical Society Rooms, Thursday, October 9, 1930. Dinner was served at 6 o'clock and at 7 o'clock the business meeting was called to order by the president, Dr. Moses Barron. Minutes of the preceding meeting were read by the secretary and approved as read. After the regular business of the evening, the scientific program was given. It is as follows:

Dr. J. M. Lajoie read a paper on "Cardiac Emergencies" (to be published in Minnesota Medicine at a later date).

Dr. W. A. Fansler followed with a report of "Two Unusual Rectal Cases"; one of "Tuberculous Intestinal Fistula," and one of "Ischiorectal Abscess with Pneumonia."

The first case I wish to report is that of a female, married, thirty-two years old. Her history is unimportant except an operation for chronic appendicitis two years ago. Two months previous to my seeing her she began to complain of pain in the rectum and had an afternoon temperature of about 99.6. Complete physical examination with *x*-ray of chest, gastro-intestinal tract, blood examiantion and tuberculin reactions were negative. The only symptoms were a rapid pulse, about 110, slight afternoon elevation of temperature and nervousness. One month after the initial onset of symptoms a small mass about 4 cm. across was felt in the left rectal wall about two inches above the ano-rectal juncture. This was opened through the rectum but did not heal and was opened more widely ten days later. She did not improve and I was asked to see her two weeks after the second operation or two months after the onset of rectal pain.

Upon proctoscopic examination nothing could be seen externally. Internally an opening could be seen in the bowel wall about two inches above the ano-rectal junction above the insertion of the levator ani muscles. A probe revealed a cavity almost two inches deep and extending downwards along the bowel wall below the level of the opening of the abscess cavity with the rectum. The appearance of the lesion gave the impression that it was probably tuberculous. Under caudal anesthesia the opening was enlarged and carried down far enough so that adequate drainage from the lowest portion of the cavity was secured and all granulations were curetted out. These were examined by University pathologist and reported as being the product of simple chronic inflammation. The lesion still did not heal as rapidly as was desired and six weeks later a more radical procedure was done with the actual cautery. The tissue was again sent to the University and a report of chronic inflammation returned. Not satisfied with this report, the remaining tissue was sent to the Glen Lake Sanatorium, where serial sections were run and definite tubercle formation found. About this time I saw an out-of-town surgeon who had operated upon her two years previous and was told that at

the time of operation he had found a tuberculous peritonitis with many miliary tubercles, the pelvis being especially involved. The patient was seen daily for almost nine months. The lesion was dressed daily and upon three occasions areas were cauterized at my office with the actual cautery. At the end of this time there was complete healing. It is quite possible that this fistula might have been healed more quickly if the incision had been carried down through both sphincters and well out into the ischiorectal fossa, thus converting the sinus into an open V-shaped cut. In doing this, however, sphincteric control would have been sacrificed and under the circumstances restoration of the sphincter by secondary operation might have been unusually difficult. I have cared for many cases of tuberculous fistulæ but I have never seen this type. To me it brought out two very interesting things: First, that a few microscopic sections, when negative, are of little value in the diagnosis of tuberculosis. In doubtful cases serial sections should be made. Second, it has again emphasized the value of the actual cautery in treating tuberculous lesions about the rectum. I use the cautery routinely in operating rectal fistulæ but its repeated use to encourage healing of sluggish areas, to me is new and the results encouraging.

The second case I wish to report is much more dramatic. I was called on the long distance phone and told that a patient was suffering from rectal pain and that he was being brought to Minneapolis that afternoon. Upon arrival his local physician gave the following history: The patient was a male, thirty-seven years old. His only previous history was that of an apical tuberculosis two years previously for which he was in a sanatorium for about three months. Since then his weight has been up and he has enjoyed good health. Two weeks previously he had injured his foot while playing golf, apparently an abrasion from his shoe. This was quite sore and a few days later he complained of pain in the rectum and some soreness in the right groin. His temperature was 101. Rectal examination revealed a tender spot just to the left of the prostate but no induration or swelling could be made out. He was kept quiet for a few days, the foot dressed and warm water enemas given. Under this he seemed to improve and returned to work. Three days later he called his local doctor at 2 a. m., because of the severe pain. The findings were the same as before except the pain in the rectum was so severe that one-fourth grain of morphine hypodermatically did not give complete relief and the temperature was again 101. Late that afternoon he was brought to me at Northwestern Hospital. When I saw him about 5 p. m., his temperature was 104.6, respirations were thirty and there was a slight degree of cyanosis. The glands in the right groin were palpable and tender. Rectal examination revealed a moderately tender area (on deep pressure) about 2 cm. in diameter and directly to the left of the upper portion of the prostate. No induration, fluctuation or other abnormality could be felt and the rectal mucosa over the tender area was normal in appearance. A blood culture was ordered. I felt certain that the symptoms were out of line with the rectal findings and asked for consultation. The leukocytes were 18,000, a moderate anemia present. *X*-ray of the chest showed a diffuse bronchial pneumonia. Blood culture revealed staphylococci and a gram-negative diplococcus in the blood stream. His condition became progressively worse and he died the morning of the fourth day after admission to the hospital.

Autopsy was obtained. The pleural cavity contained about 800 c.c. of cloudy yellowish fluid and many fresh fibrous adhesions. The pericardial sac contained fibrin threads in cloudy serous fluid. The heart was soft and flabby and contained multiple petechial hemorrhages.

The left lung showed old fibrous adhesions at the apex. Each lung showed similar findings. The pleura was covered with a thick fibrous membrane nodular in character. On section some of the nodules were seen to be infarcts; others were abscesses.

The left ischiorectal fossa contained an abscess the size of a small walnut and did not perforate the levator ani. A definite inflammatory process could be seen extending upward to the left inguinal region.

The diagnosis then was: (1) Ischiorectal abscess; (2) septicemia; (3) bronchopneumonia, bilateral with abscess formation; (4) cloudy swelling of the heart, liver, kidneys.

The history and antemortem findings in this case are to me interesting. In the first place it would seem that the original site of infection was in the foot. The second localized site where the infection struck was in the left ischiorectal fossa and inguinal glands, whether through the blood stream or lymphatics cannot be told. If through the lymphatics, then drainage of the ischiorectal fossa at the first onset of rectal symptoms might have been of benefit. At the time he was admitted to the hospital here I do not believe it would have been of value. The second interesting feature is the findings on rectal examination. The examination was absolutely negative except for the spot of tenderness. In every case I have examined previously there has been some induration or some difference in the consistency, size, shape or excursion between the normal and involved fossæ.

It is my custom to open every perirectal abscess the moment a diagnosis is made and in the past I have been able to make a diagnosis at once. In this case I was unable to tell whether an abscess was present or not and even had the patient's general condition been better I should not have felt justified in operating without further observation. This adds, then, one more point to our fund of knowledge. It may be possible to have a perirectal abscess without anatomical change demonstrable by palpation. Pain, tenderness and temperature alone may be enough to justify exploration.

DISCUSSION

DR. LAVAKE: This case is very interesting to me. We find in gynecology that unless there is the definite indication for drainage—fluctuation—we almost invariably get into difficulty if we incise. Again and again in my early practice I would be overweighed by consultants in favor of draining an indurated area. In many instances I felt convinced that it spread the infection and in may instances the incision healed and fluctuation nearby required further incision. I believe

this case was handled perfectly. The man likely had a positive blood culture when he was first seen.

DR. MCCARTNEY: Dr. Fansler's point is well taken on the point of serial sections. I do not know whether it is always possible to run serial sections; however, it depends upon the size of the piece of tissue. Some little time ago we were asked to run serial sections on a whole kidney. We figured that it would cost $3,000.00 and a girl's time for a year to do this. It is perfectly true, too, that you may get the thing you are looking for in the first section, and then again you may not. We get pieces of tissue every now and again, where they are thinking of tumor, and yet multiple sections do not show any sign of the disease condition present in the piece submitted.

DR. FANSLER (closing): I do not believe that draining this abscess would have made any difference in the outcome. Possibly if I had seen him two or three days earlier, I might have felt justified in exploring the ischiorectal fossa. If I had seen him before his pulmonary symptoms developed and had drained the abscess, it is still a question in my mind as to whether it might have prevented the pneumonia for most likely the blood stream infection was already present.

In answer to Dr. Wynne's question as to whether a subcutaneous tuberculin test had been done. I questioned the doctor especially about this. I have seen a large number of tuberculous fistulæ, and I was suspicious about this one. He said he had done a tuberculin test, rayed the chest and had all other laboratory data and could not find a thing.

In answer to Dr. Barron's question as to whether the doctor had watched the patient over a period of time. He said he had observed and examined this patient repeatedly, including numerous rectal examinations. I may also add that this physician is a very careful, keen and competent clinician. I believe the infection probably originated in the pelvis. We do know that tuberculous abscesses are almost painless to touch unless complicated by pyogenic organisms. This one could easily have developed painlessly and was in the beginning deeply enough seated that it could not be palpated.

DR. DONALD MCCARTHY presented two cases, (1) "Malignant Hypertension," and (2) "Diabetes with Substernal Thyroid."

The meeting adjourned at 8:30 P. M.

H. BRIGHT DORNBLASER, M. D.,
Secretary.

Meeting of November 13, 1930

The regular monthly meeting of the Minneapolis Clinical Club was held on Thursday evening, November 13, 1930, in the Lounge of the Hennepin County Medical Society. Dinner was served at 6 o'clock and the meeting was called to order shortly after 7 o'clock by the President, Dr. Moses Barron.

There were 28 members and 2 visitors present.

Minutes of the October meeting were read and approved.

After a short business meeting, the scientific program was taken up as follows:

DR. A. A. ZIEROLD reported a case of "Sympathectomy as a Cure for Angina Pectoris," and after the reading of the history the patient was presented to the Society. The case report is as follows:

This patient is a man 60 years of age who has been suffering for three years from anginal attacks of increasing severity and frequency. He has been under the care and observation of an internist and the clinical diagnosis of angina pectoris has been confirmed by repeated electrocardiograms. During the past few months the pain has been sufficient to require large doses of morphin regularly. As the patient exhibited no evidence of cardiac decompensation it was thought that, could he be relieved of pain, he might be restored to some degree of activity and usefulness.

It was decided to attempt the alcohol injection of the cervical and thoracic sympathetic ganglia as advocated by Mixter and White. Because of the deep layer of fat and muscle overlying the upper thoracic spine the original technic could not be followed. Under novocain infiltration anesthesia a five-inch incision was made caudally from the level of the seventh cervical spine, parallel to and two inches distant from the upper dorsal spinous processes. The incision was carried through the muscles to the ribs, exposing them at the articulations with the transverse processes. Beginning with the first rib, a needle was introduced under the lower border at this point and directed medially and upward to reach the site of the sympathetic ganglion without entering the pleura. It was then aspirated to confirm its location, and a few c.c. of 1 per cent novocain was injected. Five needles were placed after this manner under the borders of the upper five ribs. In each of these 5 c.c. of 80 per cent alcohol were injected. The patient experienced no pain or discomfort, but noted an anesthesia of the inner side of the left arm and the left chest wall corresponding to the distribution of the upper five thoracic nerves. On the following day this was replaced by a similar area of hyperesthesia which gradually disappeared in the following two weeks.

The patient's convalescence was uneventful. The wound healed promptly and in a normal manner. It is now four weeks since his operation and he has not experienced a single anginal pain.

You will note that as evidence of the interruption of the splanchnic pathways, the patient has the enophthalmos and constricted pupil of a Horner's syndrome. The left hand is also warmer than the right as a result of vasoconstrictor loss.

It is yet too early to warrant any positive statement, but it appears reasonable to believe that the patient will be free from pain for some time, if not permanently.

DISCUSSION

DR. H. L. ULRICH: I am sorry I did not know this patient was coming this evening and I would have looked over my notes before the meeting. I saw this man in consultation three years ago. He had a distinct coronary closure from which he recovered and went about his work again. This year I saw him again and at that time he was in a state of persistent anginal attacks. I suggested that he be operated on and that the thoracic ganglia be injected. This is as far as I am acquainted with the patient. At the time I saw

him first he had distinct coronary disease and now gives evidence of conorary disease in the electrocardiogram. He is entirely free from pain.

DR. ARCHIE H. BEARD: I understand that the results of this injection have the tendency to disappear, and that in time the attacks of angina are apt to return. I have the impression that White's last report stated that at the end of about three years some were having anginal attacks.

Do you think that this was due to faulty technic at the time of the injection, and do you know whether the same results occur when you cut down the ganglia and inject them direct?

DR. ZIEROLD: It is probable that the percentage of unsatisfactory cases is in direct proportion to those which do not develop a Horner's syndrome as secondary evidence of successful injection.

DR. J. C. MICHAEL: Do you attempt to expose the ganglia?

DR. ZIEROLD: No.

DR. MICHAEL: One would not, of course, expect a return of function in those sympathetics?

DR. ZIEROLD: I assume that the ganglia will be destroyed or irreparably injured by the alcohol.

DR. J. S. MCCARTNEY read a paper on "Cirrhosis of the Liver," illustrating it with numerous lantern slides. (Paper to be published later, probably in the Archives of Internal Medicine.)

DISCUSSION

DR. H. L. ULRICH: I merely wish to emphasize that clinically I have been struck by the fact that in adults about 40 years of age, or over, when you find a pleural effusion that you cannot explain, it is well to look at the liver to see whether or not that patient has cirrhosis. The pleura may be the first cavity to show fluid.

The liver is a strange field, a strange organ. In the last few years we are getting reports that by injecting a pigment into one group of portal veins it comes up in one lobe of the liver, and by injecting another group it shows up on the other side of the liver. This differentiation of the blood in the portal vein is very strange. That is just one of the little mysterious points about the liver. And these plates we have seen tonight are not made any clearer by such knowledge, but it merely shows why they may be so confusing.

Great credit is due to Dr. McCartney for his persistent attempt to bring clearness to this subject. We should be very grateful to him for bringing so much order out of an apparent chaos.

DR. BARRON: This is the type of paper that can bear a great deal of discussion both from the clinical and pathological standpoints.

DR. FREDERICK H. SCHAAF gave a talk on "Hunting Big Game with a Movie Camera," and showed two reels of pictures taken on his recent hunting trip in Canada. This was a very enjoyable addition to the program.

The meeting adjourned.

H. BRIGHT DORNBLASER, M.D.,
Secretary.

NEW AND NON-OFFICIAL REMEDIES

The Council of Pharmacy has accepted the following articles:

ABBOTT LABORATORIES
- Gold Sodium Thiosulphate-Abbott

ELI LILLY & CO.
- Erysipelas Antistreptococcic Serum-Lilly (Concentrated Globulin)

MEDICAL ARTS LABORATORY, INC.
- Antirabic Vaccine, Semple Method

NATIONAL DRUG CO.
- Antipneumococcic Serum, Type I
- Ointment Scarlet Red Biebrich 8 Per Cent
- Typhoid-Paratyphoid A Vaccine

PARKE, DAVIS & CO.
- Ventriculin

RICHARDS PHARMACAL CO., INC.
- Richards Psyllium Seed

E. R. SQUIBB & SONS
- Diphtheria Toxoid-Squibb, twenty 1 c.c. ampule packages
- Diphtheria Toxoid-Squibb, two 1 c.c. ampule packages
- Normal Horse Serum, one 50 c.c. vial package
- Ragweed Pollen Allergen Solution-Squibb (3 vial treatment package)
- Timothy Pollen Allergen Solution-Squibb (3 vial treatment package)

TRUTH ABOUT MEDICINES

The following products have been accepted by the Council on Pharmacy and Chemistry of the American Medical Association for inclusion in New and Non-official Remedies:

McKesson's Vitamin Concentrate of Cod Liver Oil.—A cod liver oil concentrate having not less than eleven times the minimum Vitamin A potency of cod liver oil, U.S.P., and not less than eleven times the Vitamin D potency of a potent cod liver oil used as a standard. The product possesses properties similar to those of cod liver oil so far as these depend on the fat-soluble vitamin content of the latter. McKesson and Robbins, Inc., Bridgeport, Conn. (Jour. A. M. A., November 1, 1930, p. 1347.)

Maltine with Cod Liver Oil and Iron Iodide.—This product is composed of maltine, 70 per cent; cod liver oil, 30 per cent; and ferrous iodide, 0.44 Gm. per hundred c.c. (2 grains to each fluid ounce). Maltine is a preparation essentially similar to extract of malt, U.S.P., but it contains 3.8 per cent of alcohol, is prepared from malted barley, oats and wheat; its Vitamin B_1 and B_2 content and its starch converting power are controlled by assay. Maltine with cod liver oil and iron iodide contains in 100 c.c. from 23,000 to 25,000 Vitamin A units as determined by the U. S. Pharmacopeia assay and its Vitamin D potency is controlled by assay. Maltine Company, Brooklyn, N. Y.

Diphtheria Toxoid.—A diphtheria toxoid (New and

Non-official Remedies, 1930, p. 364), prepared from diphtheria toxin of which the L+ dose is 0.2 c.c. or less. The toxin is treated with formaldehyde and is tested for antigenic power. The finished product is adjusted to contain in 2 c.c. enough of the toxoid for one immunization treatment. It is marketed in packages of one immunization treatment. Lederle Laboratories, Inc., Pearl River, N. Y. (Jour. A. M. A., November 15, 1930, p. 1505).

Ampoule Sterile Solution Dextrose, U.S.P., 5 Gm., 10 c.c.—Each ampule contains dextrose, U.S.P., 5 Gm., in distilled water to make 10 c.c. E. S. Miller Laboratories, Inc., Los Angeles.

Ampoule Sterile Solution Dextrose, U.S.P., 10 Gm., 20 c.c.—Each ampule contains dextrose, U.S.P., 10 Gm., in distilled water to make 20 c.c. E. S. Miller Laboratories, Inc., Los Angeles.

Ampoule Sterile Solution Dextrose, U.S.P., 25 Gm., 50 c.c.—Each ampule contains dextrose, U.S.P., 25 Gm., in distilled water to make 50 c.c. E. S. Miller Laboratories, Inc., Los Angeles.

Dextrose (d-Glucose) Unbuffered and Without Preservative, 10 Gm., 20 c.c. Ampule.—Each ampule contains dextrose, U.S.P., 10 Gm., in distilled water to make 20 c.c. H. K. Mulford Co., Philadelphia.

Dextrose (d-Glucose) Unbuffered and Without Preservative, 25 Gm., 50 c.c. Ampule.—Each ampule contains dextrose, U.S.P., 25 Gm., in distilled water to make 50 c.c.; accompanied by an ampule of 2 c.c. of a buffer solution. H. K. Mulford Co., Philadelphia.

Dextrose (d-Glucose) Unbuffered and Without Preservative, 25 Gm., 50 c.c. Double End Vial.—Each double end vial contains dextrose, U.S.P., 25 Gm., in distilled water to make 50 c.c.; accompanied by an ampule of 2 c.c. of a buffer solution. H. K. Mulford Co., Philadelphia.

Dextrose (d-Glucose) Unbuffered and Without Preservative, 50 Gm., 100 c.c. Ampule.—Each ampule contains dextrose, U.S.P., 50 Gm., in distilled water to make 100 c.c.; accompanied by an ampule of 4 c.c. of a buffer solution. H. K. Mulford Co., Philadelphia.

Ventriculin.—Desiccated, defatted, hog stomach.—It is assayed clinically by observation of the reticulocyte response, the standard being an increase of red blood cells at the rate of about one hundred thousand cells per cubic millimeter per week when the product is administered to patients suffering from pernicious anemia. Stomach tissue of animals has been shown to contain a principle capable of stimulating the bone marrow to form immature red cells in large numbers. When, during the first ten to fifteen days treatment with ventriculin, a satisfactory rise in reticulocytes occurs, this is evidence that effective and progressive blood regeneration is taking place. Ventriculin is supplied in the form of a powder in vials containing 10 Gm. Parke, Davis & Co., Detroit. (Jour. A. M. A., November 22, 1930, p. 1589.)

Synephrin—Hydroxyphenylmethylaminoethanol Hydrochloride.—The hydrochloride of an alkaloid obtained synthetically. Synephrin is used as a vasoconstrictor. It is less toxic than either epinephrine or ephedrine, and its vasoconstrictor action, while not so pronounced as that of epinephrine, endures for a longer time. In combination with procaine hydrochloride it is useful for local anesthesia in dental operations and in minor surgery in cases in which a bloodless area is not required. The drug is also supplied in the form of Synephrin Solution "A," Ampoules Synephrin-Procaine, 3 c.c., and Hypodermic Tablets Synephrin-Procaine. Frederick Stearns & Co., Detroit.

Mead's Dextri-Maltose with Vitamin B.—A mixture containing approximately: maltose, 52.58 per cent; dextrine, 39.80 per cent; protein, 4.34 per cent; mineral salts, 2.28 per cent; and moisture, 1.00 per cent. It is standardized physiologically to contain in each 2.5 Gm. the Vitamin B_1 and B_2 equivalent of approximately 1 Gm. of dried yeast or 2 Gm. of wheat embryo. Mead's Dextri-Maltose with Vitamin B is proposed for use in the diet of infants suffering from Vitamin B deficiency. Mead Johnson & Co., Evansville, Ind. (Jour. A. M. A., May 3, 1930, p. 1405.)

Scillaren.—A mixture of the natural glucosides, scillaren-A and scillaren-B, occurring in fresh squill *Urginea maritima*, in the proportions in which they exist in the fresh crude drug; namely, about 2 parts of scillaren-A to 1 part of scillaren-B. Completely dried scillaren contains approximately 98 per cent of the active glucosides. Scillaren dried in a high vacuum at 78° C. for fifteen hours loses not more than 6 per cent of its weight. The cardiac action of scillaren is essentially similar to that of digitalis; but this action is apparently less persistent than that of digitalis. Scillaren is administered orally and is supplied in the form of tablets containing 0.8 mg. (1/80 grain) of scillaren and in the form of a solution containing 0.8 mg. (1/80 grain) of scillaren. Sandoz Chemical Works, Inc., New York.

Parke, Davis & Company's Codliver Oil with Viosterol 5 D.—A brand of codliver oil with viosterol 5 D.-N.N.R. (New and Non-official Remedies, 1930, p. 257). Parke, Davis & Co., Detroit. (Jour. A. M. A., May 31, 1930, p. 1761.)

CONSULTATION BUREAU

(Continued from page 92)

Answer.—If abortion is inevitable and there are strong pains without bleeding, the patient should be put to bed and nature allowed to take its course. If there is no possibility of avoiding the termination of pregnancy, and you are absolutely sure about this, and the process is lagging, stimulation of the uterus with quinine or pituitrin is indicated. If the patient is bleeding profusely a sterile pack may be inserted and removed before twelve hours. If the bleeding is excessive and cannot be controlled by a pack, the cervix should be dilated with a sponge forceps. In any event the patient should be kept in bed from five to seven days after delivery to allow involution to take place. Curettage should never be done even if the patient continues to bleed and material is present in the uterus until she has been free of fever for ten days and has a normal leukocyte count. If the patient is still having trouble (bleeding) and has been free of fever for ten days and has a normal leukocyte count, then a sponge forceps (sterile) should be introduced into the uterus with great precaution to avoid infection, and anything which is loose should be gently taken out.

PROGRESS

Abstracts to be submitted to Section Supervisors.

Members are urged to abstract valuable articles which they run across in their reading and send the abstracts to the physicians in charge of the respective sections. In order to avoid duplication it would be well to communicate with one of the section supervisors before the article is abstracted.

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EYE, EAR, NOSE AND THROAT

ORAL INFECTIONS AND THEIR RELATION TO DISEASES OF THE EYE: Henry Dunning (Arch. Ophthal., Sept., 1930, p. 315). Intestinal bacteria have a predilection for the pulp of teeth in which vitality is low. They find their way to the pulp of the teeth through the blood stream and cause the death of the teeth by their action.

Teeth become infected most often in the following three ways: (1) By the intradental route, in which the decay involves the pulp, then thepulp canal and goes onward through the apex of the tooth. This causes the true apical infection. (2) By the peridental route under the gingivæ, in which the peridental attachment into the alveolus is stripped up, thereby allowing bacteria to gain access to the bone. (3) Through the blood stream.

Pus under pressure, locked in a spongy vascular bone, gives no trouble for years, but continuously and relentlessly breaks down the patient's resistance until the chemistry of the body has undergone certain changes. Secondary changes then take place in the blood and perhaps all other tissues, including the eye.

For culturing organisms the tooth must be extracted under local anesthesia, as general anesthesia allows the socket to become contaminated with the increased flow of saliva and increased bleeding of the adjacent parts.

The question of pulpless teeth has not been settled as yet by the dental profession although the prevailing opinion is that nearly all pulpless teeth are infected and that most of them can not be made safe for the patients who are carrying them. Most pulpless teeth ought to be removed in a patient of about 50 years of age in order to reduce to a minimum the number of virulent bacteria, with their toxins, which are present in the body, just as it is a correct procedure to cut down to a minimum the number of bacteria in the milk or other food that one is to consume.

Furthermore it is poor practice to curet infected alveoli of teeth after extraction as it has taken many months to build up a barrier around the tooth and this should not be broken down, allowing the organism at the apex of the tooth to have free access into the walls of the newly formed wound. It is possible by this procedure to let loose into the system many dangerous bacteria and their toxins.

Carefully observed cases of orbital abscess, iritis, and uveitis that can be definitely ascribed to dental sepsis, have been recognized and described. While the exact process of extension is not known, yet the improvement of these eye conditions following the correction of the disease of the teeth is sufficient evidence that a close relationship exists.

The mode of transmission is probably in one of these three ways:

(1) Direct extension through bone.

(2) Direct extension along the periosteum of the malar bone.

(3) Blood stream (the most important).

The literature and case reports repeat over and over the evidence that infections of the mouth play an important rôle in connection with ophthalmic disease.

GERALD M. KOEPCKE, M.D.

PEDIATRICS

DEFECT OF THE DICK TEST AND ITS REMEDY: T. Toyoda and Y. Futagi (Jour. Infect. Diseases, 46-196, March, 1930). Toyoda and Futagi conclude that the Dick toxin consists of two toxins one of which is heat-labile and the other is heat-stable. The heat-labile toxin, which they consider to cause the symptoms of scarlet fever in its early stage, contains no nitrogen, and is easily destroyed in one half hour at 80° C. This heat-labile component of the Dick toxin gives strongly positive cutaneous reactions in individuals who are not immune to scarlet fever, and never

causes allergic reactions. The heat-stable toxin, on the contrary, is not destroyed in two hours at 100° C., and when employed in skin tests often produces allergic reactions in those immunized against scarlet fever, which may account for the failure of the studies of various investigators to coincide with one another. Toyoda and Futagi feel, therefore, that in performing Dick tests one should use only the pure heat-labile toxin, and that skin reaction to this toxin accurately indicates susceptibility or immunity to scarlet fever.

C. A. STEWART, M.D.

CHOREA: A. E. Vipond, M.D., Montreal (Arch. of Ped., October, 1930, Vol. XLVII, No. 10). Chorea is due to a toxemia of the brain and spinal cord. This produces an irritability of the higher centers, including the intellectual centers, the cortical centers, the nuclei, which control voluntary movements, including the corpus of Luys, the red nucleus, the substantia nigra, and the lenticular nucleus.

It is pleasant to see all of the symptoms disappear under the sterilized milk treatment. He believes that this form of treatment is the best method of treating chorea in children. Method of injecting the sterilized milk: In injecting the milk, use a fair sized needle, as a small one will get blocked; inject five c.c. of sterilized milk every three days. Some patients will only require four to five injections, while others, mild cases, may require 18 to 20 injections to effect a cure.

How is it that sterilized milk injections effect a cure in chorea? Within one hour's time after the injection, the W. B. C. increase to an astonishing number. From the normal 8,000 we get an increase of from 11,000 to over 24,000, 16,000 being a common number. They prevent toxemia and then a cure takes place. They likely do away with the micro-organisms which produce these toxins.

The control centers, the intellectual centers and the mid-brain centers regain their tone and the patient's nervous system runs in a normal groove again.

R. N. ANDREWS, M.D.

SUSPECTED JUVENILE TUBERCULOSIS: Frederick Eberson, Ph.D., M.D., Jessie P. Delprat, M.D., and Ernst Wolff, M.D., San Francisco (Amer. Jour. of Diseases of Children, October, 1930, Vol. 40, No. 4). The symptoms that occurred the great number of times in both groups (group 1, giving positive tuberculosis, and group 2, negative) were cough, nervousness, fatigue, and lack of appetite. It was found that in group 1, 11.2 per cent, and in group 2, 12 per cent presented no clinical observations in regard to tuberculosis. This study has brought out the important fact that there was little or no difference between a group with positive and a group with negative tuberculin reactions, as judged by nine symptoms and ten clinical signs under consideration.

Two groups of children were selected on the basis of their reaction to tuberculin. It was found that in the positive group (206 patients), 36.4 per cent presented no symptoms, as compared with 42 per cent among the negative reactors (100 patients).

The differences between the percentages of total occurrences of symptoms in both groups were never greater than 7 per cent, except in the case of cough, which in the series of persons with negative reactions exceeded those with positive reactions by 13.4 per cent.

In the positive group, malnutrition, dullness of the manubrium sterni, and enlargement of the cervical lymph nodes, occurred more often; while D'Espine's sign, paravertebral dullness, and auscultation exceeded among those with negative reactions. It was found that in the positive group 5.8 per cent, and in the negative group 15 per cent presented no roentgen evidence. In the positive group, hilus calcifications, increased bronchial markings, and thickened interlobar pleura, occurred more frequently; in the negative group, peribronchial infiltration occurred more often. Observations that have been considered in the past as pathognomonic of tuberculosis were associated with nontuberculous processes in the lung. Such roentgen evidence, occurring alone, appeared to have no diagnostic value.

Evidence that has been presented in this study showed that commonly accepted clinical symptoms and signs have no diagnostic significance unless these are definitely correlated with positive tuberculin tests and roentgen observations that are positive for tuberculosis.

In terms of these more modern concepts an explanation has been found for the infrequent pathologic evidence of tuberculosis at autopsy as compared with the diagnoses on the basis of *x*-ray films of the chest.

R. N. ANDREWS, M.D.

THE USE OF POWDERED MILK AND MILK DERIVATIVES IN THE CONCENTRATION OF INFANT FOODS: Arthur F. Abt, M.D., Chicago, Ill., and Benjamin F. Feingold, M.D., Glencoe, Ill. (Arch. of Ped., October, 1930, Vol. XLVII, No. 10). Nearly all underweight and also many normal weight infants have a much greater nutritional requirement than the 40-50 calories per pound of weight which is considered the average normal need. In the premature, the feeble new-born, the poor nurser, or in all infants whose capacity is so limited that the average 24-hour food requirement can not be met by the usual formulæ, it becomes imperative to concentrate the feeding. It is a too prevalent theory that dystrophic infants must be fed on dilute mixtures.

One of the first methods for concentrating infant formulæ was the addition of cream. Later cereal foodstuffs were combined with the mixtures. Malt soups and butter-flour mixtures were employed. More recently stronger milk mixtures (⅔ or ¾ milk) have been used. Concentrated protein milk and finally whole milk with very high percentages of carbohydrate, such as Schick's Dubo with 17 per cent sugar, have been advised. Unless the infant receiving a high carbohydrate formula was kept under closest observation, diarrhea easily developed. The author has found that the simple addition of powdered milk and milk derivatives can solve the problem of concentrating formulæ in a very practical and satisfactory manner.

The author has used calcium caseinate for breast-fed infants developing diarrhea. In these cases the calcium caseinate is added to water in the proportion of one level tablespoonful of the powder to three ounces of water. Occasionally we encounter a newborn infant with small, frothy stools accompanied by a progressive gain in weight. By preceding the breast feeding with one ounce of powdered protein milk mixture and limiting the time at breast to 10 minutes, we have been able to check the frequency of stools and readily clear up the sore buttocks. With prematures having normal stools, the addition to one to two drams of condensed milk to the whole day's feeding of breast milk will sufficiently increase the caloric intake to produce a progressive rise in the weight curve. Very often a normal child is encountered, who is not contented with the caloric intake offered by a formulæ prescribing such a volume, while on the other hand the baby is unable to care for a larger amount. In such instances, add condensed milk or powdered whole milk. The powdered milks and their derivatives also find a very useful rôle in the treatment of vomiting in infancy and childhood.

R. N. ANDREWS, M.D.

ROENTGENOLOGY

THE IRRITABLE COLON: John Muir, M.B. (Brit. Jour. of Rad., III, 33, Sept., 1930, p. 391). Many vague dyspeptic symptoms are associated with colonic disorders in which treatment of the direct gastric symptoms has proved only palliative. The author calls this relationship the "radiological symptoms complex," and it is present in cases in which the duodenum shows signs of irritation which may be due to local ulcer but no direct evidence of such ulcer is detected and the appearance may be due to reflex irritation as from a pathological gallbladder, appendix or colon. This relationship to pathological gallbladder and appendix has been noted frequently.

The irritable colon is the third relationship and these cases are marked by spasm of the pyloric vestibule, pyloris, and duodenal bulb of varying severity and location. There is tenderness on palpation over the colon often accentuated over the middle of the transverse colon where the site of its origin may be mistaken as being in the lower pole of the stomach, or at other times at its maximum over the descending colon. The irritable colon shows exaggerated and irregular segmentation or haustration eighteen to twenty-four hours after the opaque meal. At times there is also a definite narrowing without segmentation especially in the splenic flexure and descending colon which is generally better shown by the opaque enema. Here the radioscopic observation is very essential. The most information is obtained by a combination of the opaque meal and enema. The condition can generally be differentiated without difficulty from an organic infiltration by the permanency of the defect in the latter.

Some cases present localizing symptoms such as indefinite backache, cramp-like abdominal pains which direct attention to the colon, chronic constipation or occasional or persistent diarrhea. Apart from these, the most common case is one which presents symptoms usually associated with or ascribed to duodenal ulcer.

In conculsion, the author says that this study is an endeavor to interpret function by radiological means.

JACOB SAGEL, M.D.

THE PATHOLOGY OF EARLY PULMONARY TUBERCULOSIS AS REVEALED BY X-RAYS: Peter Kerley (Brit. Jour. of Rad., III, No. 33, Sept., 1930, p. 404). The author discusses the earliest tuberculous focus in regard to its anatomy, situation and mode of entry of the bacillus. According to *x*-ray appearance, there are three distinct types of infantile pulmonary tuberculosis: (1) tuberculous bronchopneumonia, (2) localized caseous pneumonia, (3) diffuse caseous pneumonia. All three types present a visible pulmonary lesion, and in about fifty per cent enlarged bronchial glands can also be demonstrated. *X*-ray evidence supports the view of Ghon, McCallum and Küss that the primary infantile lesion is pulmonary and not glandular. The development of the three types is traced and the *x*-ray appearance noted.

In adults, the infraclavicular region is the commonest location, the right side being more frequent than the left. In 1922, Assman described a round sharply defined opacity in the right subclavicular region which he called a primary tuberculous focus, and which he believed was a common method of occurrence of tuberculosis. Two such case at autopsy showed a localized caseous pneumonia deep in the middle of the upper lobe where no physical signs would be possible. The lesion is identical in infants and adults, and differs only in location. For mechanical reasons, the base of the lung is attacked in children and the upper lobe in adults.

The prognosis in early cases depends largely on the inherent resistance of the patient, and also on the site of the lesion. With reasonable care most infants cure themselves. The early adult focus may heal by resorption or by calcification and fibrosis. The constant situation of the lesion in the infraclavicular region points more to reinfection rather than a recrudescence of an old infection. Large caseous pneumonias in adults are very dangerous, and tend to end fatally in a short time. This lesion is the "galloping consumption" of the last century.

A triangular caseous pneumonia in an adult appears to spread and break down more quickly than the round caseous pneumonia of Assman. Pure apical tuberculosis tends to heal spontaneously, and the prognosis is always good. This has been proved by Fishberg in a large series of cases.

JACOB SAGEL, M.D.

BOOK REVIEWS

Books listed here become the property of the Ramsey and Hennepin County Medical libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

BOOKS RECEIVED FOR REVIEW

MENTAL ASPECTS OF STAMMERING. C. S. Bluemel, M.A., M.D., L.R.C.P. (Lond.), M.R.D.S. (Eng.). Clinical Instructor in Neurology of Colorado School of Medicine, etc. Illus. 152 pages. Price, $2.50. Baltimore, Md.: The Williams & Wilkins Company.

EDWARD JENNER AND THE DISCOVERY OF SMALLPOX VACCINATION. Louis H. Roddis, M.D. 155 pages. Price $1.00. Banta Publishing Company, 1930.

This interesting little volume will afford an enjoyable evening's reading to any one. It includes references to the prevalence of smallpox in the earliest historical records, and describes the development of inoculation with smallpox. This practice, designed to infect susceptible individuals with mild smallpox, was practiced in the Orient during the seventeenth and eighteenth centuries. This measure operated with considerable success, although occasionally fatal cases developed in inoculated persons; yet it was infinitely preferable to the malignant smallpox, epidemics of which ravaged Europe frequently. Inoculation with human smallpox was popularized in Europe early in the eighteenth century by Lady Mary Montague, who observed the practice in Constantinople. She writes in the skeptical vein adopted by quacks of the present era: "I am patriot enough to take pains to bring this useful invention into fashion in England; and I shall not fail to write to some of our doctors very particularly about it, if I knew any one of them had virtue enough to destroy such a considerable branch of their revenue for the good of mankind."

In America, inoculation was warmly urged by Benjamin Franklin, and through his influence it attained considerable vogue.

Jenner's life is pictured in a readable manner. His medical career began at the age of thirteen, as a surgeon's apprentice. In his twentieth year he became a house pupil of the renowned surgeon and anatomist, John Hunter, and two years later took up rural practice in Gloucestershire. Jenner's broad interests in biology and natural history caused him to investigate thoroughly the common belief among the laity that infection with cowpox, such as frequently occurred among dairy-hands, invariably made them immune to smallpox. His memorable application of this fact in scientific manner, and his demonstration of the result by deliberately exposing those vaccinated to smallpox later, without infection developing, won immediate recognition in England, and imperishable fame to Jenner. In but a few years vaccination was practiced generally over the civilized world. In 1799 it was introduced in America by Dr. Benjamin Waterhouse of Boston, who obtained virus from Jenner, and vaccinated his own seven children. Later he tested its efficacy by exposing three of them to smallpox, with no ill effects.

Jenner's versatility and wide interests manifested themselves by his researches in biology and ornithology; he was a poet of no mean caliber, and a keen student of human nature. The latter quality reveals itself in the following passage, taken from a letter wherein he comments on the fact that the boyish appearance of his capable assistant interfered with his gaining the confidence of his patients: "For it is by appearance, not from real knowledge of things, that the world (at least a major part of it) forms a judgment. A look of significance, a peculiar habit, and a very scanty acquaintance with the human machine, will make a man pass current for a great physician."

THOMAS MYERS, M.D.

ALIMENTARY ANAPHYLAXIS (gastro-intestinal food allergy). Guy Laroche, Charles Richet and François Saint-Girons. 139 pages. Trans. by Mildred P. Rowe and Albert H. Rowe. Berkeley, Calif., Univ. of Calif. pr., 1930.

This is a small monograph which was written first in 1920 in support of an argument at that time little held, *i.e.*, the occurrence of sensibility and anaphylaxis from alimentary absorption of foreign proteins. It has been translated into English and reprinted with footnotes by the authors written this year from the knowledge gained in the last ten years. While the information contained is now more or less commonplace, this volume is worth reading partly as a summary of the knowledge of alimentary anaphylaxis, and partly to see the progress of ten years. There is much left out, of course, but an evening spent on "Alimentary Anaphylaxis" will be well spent.

H. B. SWEETSER, JR., M.D.

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